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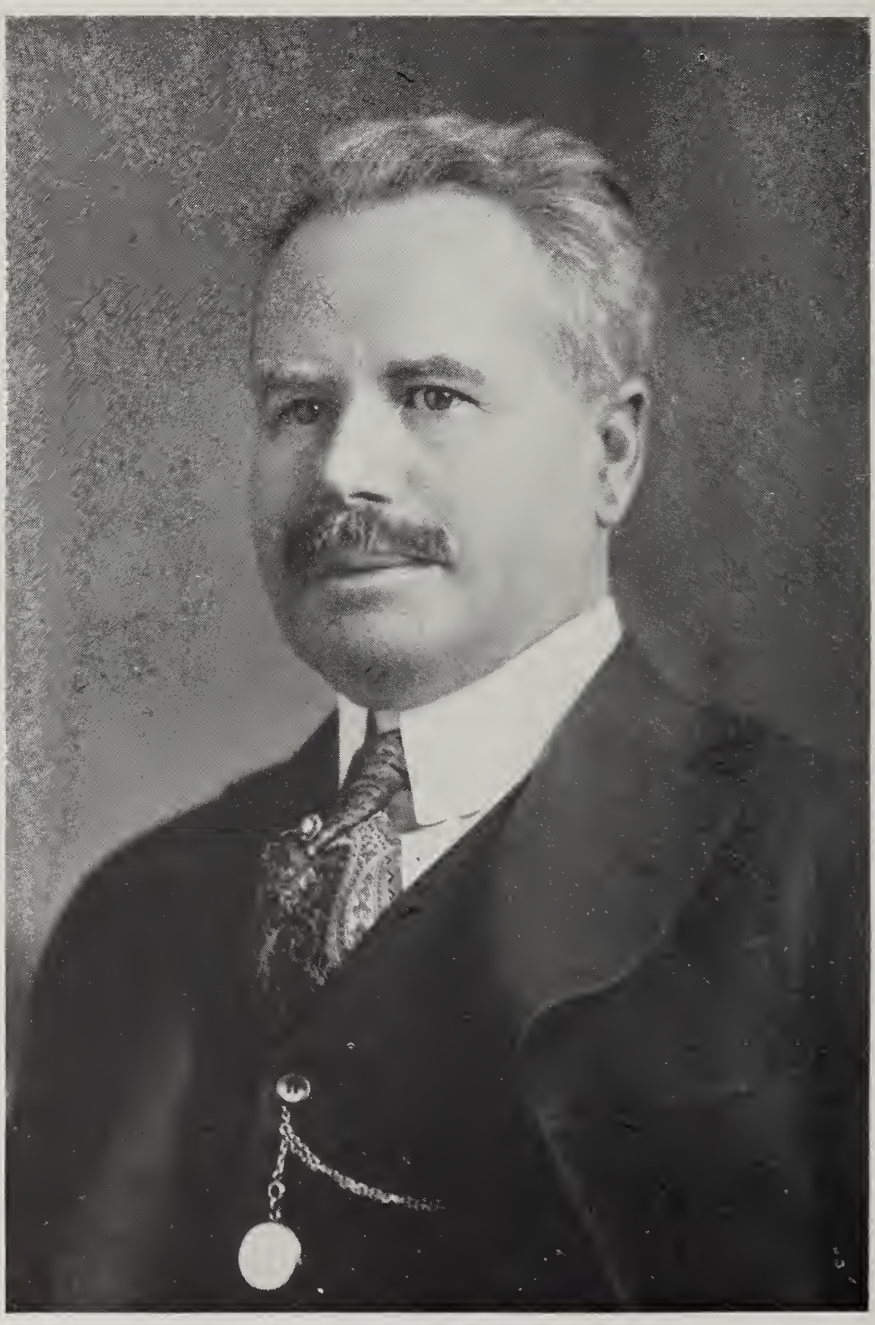

BYRON BENNETT DAVIS

PRESIDENT 1914

TWENTY-FOURTH ANNUAL MEETING

TRANSACTIONS

OF THE

WESTERN SURGICAL ASSOCIATION

Twenty-Fourth Annual Meeting

Denver, Colorado, December 18 and 19, 1914

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Oliver J. Fay, M. D. - - - Des Moines

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EX-PRESIDENTS AND MEETING PLACES

PRESIDENT	PLACE	YEAR
S. S. Todd*	Topeka	1891
Milo B. Ward*	Kansas City	1892
Milo B. Ward*	Des Moines	1893
Lewis Schooler	Omaha	1894
John E. Summers, Jr.	Kansas City	1895
Thomas J. Beattie	Topeka	1896
Joseph Eastman*	Denver	1897
David S. Fairchild	Omaha	1898
Homer C. Crowell	Des Moines	1899
O. Beverly Campbell	Minneapolis	1900
August F. Jonas	Chicago	1901
James E. Moore	St. Joseph	1902
Alex. Hugh Ferguson	Denver	1903
Charles H. Mayo	Milwaukee	1904
Harvey D. Niles*	Kansas City	1905
Malcolm L. Harris	Salt Lake City	1906
Charles W. Oviatt*	St. Louis	1907
William W. Grant	Minneapolis	1908
Arthur L. Wright*	Omaha	1909
John P. Lord	Chicago	1910
Amos W. Abbott	Kansas City	1911
Lewis L. McArthur	Cincinnati	1912
Jabez N. Jackson	St. Louis	1913
Byron B. Davis	Denver	1914

*Deceased.

The Western Surgical Association does not hold itself responsible for, or necessarily endorse, any of the papers printed herein.

ARTHUR T. MANN, Secretary-Treasurer.

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Executive Session

December 18, 1914—First Day—Morning Session

The Association meet at the Brown Palace Hotel, Denver, Colorado, at 9:00 a. m., and was called to order by the President, Dr. Byron B. Davis, of Omaha, Nebraska.

Dr. David W. Basham, of Wichita, read a paper entitled, "Report of a Case of Post-operative Anuria Occurring Nearly Two Weeks After Nephrectomy for Hypernephroma, with the Therapeutics of the Case."

The paper was discussed by Drs. Charles D. Lockwood, James F. Percy, and Jabez N. Jackson.

Dr. Joseph R. Eastman, of Indianapolis, read a paper entitled, "Permanent Compression of Both Carotids in General Idiopathic Epilepsy."

The paper was discussed by Drs. John P. Lord and Emil H. Beckman.

Dr. Emil H. Beckman read a paper entitled, "Recovery from Paralysis Following Decompression of the Spinal Cord." The paper was discussed by Dr. William W. Grant and by Dr. George A. Moleen, of Denver, present by invitation.

Dr. Charles D. Lockwood read a paper entitled, "Factors Determining Mortality in Prostatectomy." The paper was discussed by Dr. S. C. Beede, of David City.

Dr. Donald Macrae, of Council Bluffs, read a paper entitled, "Gastrodiaphragmatic Attachment of Tubal Pregnancy."

On motion, the Association adjourned until 2:00 P. M.

First Day—Afternoon Session

The Association reassembled at 2:00 P. M., and was called to order by the President, Dr. Byron B. Davis, who said:

I wish to take this opportunity to thank the Association for the honor conferred upon me, and to express to you my appreciation. I know of no higher honor that could come to a surgeon than to preside over such an Association as this.

For my annual address I have not taken up any one topic, but I have touched upon three different subjects that seem to me of pretty general interest to surgeons.

The President's address was then delivered by him with Dr. Leonard Freeman, of Denver, First Vice-President, in the chair.

At the close of the address, Dr. Freeman said: "Is there anything in this interesting and instructive address which the Association wishes to take action on on any way?

Dr. Horace G. Wetherill, of Denver, said: "May I suggest that it might be a good plan to refer the address to a committee for such recommendations as may seem appropriate and proper under the circumstances.

Dr. Freeman: Did you make that as a motion?

Dr. Wetherill: I am willing to make it as a motion; yes, Sir.

The motion carried without dissent, and Dr. Freeman announced that the committee would be named later. At a later hour the following members were announced on this committee: Drs. H. G. Wetherill, Joseph R. Eastman, and E. P. Quain.

Dr. Byron B. Davis returned to the chair.

Dr. Frank G. Nifong, of Columbia, then read a paper entitled, "Some Observations on Mesenteric Occlusion." The paper was discussed by Drs. A. A. Law, A. E. Benjamin, A. E. Hertzler, and F. A. Dunsmoor.

Dr. John P. Lord read a paper entitled, "Operation to Prevent Recurrence of Intussusception." The paper was discussed by Drs. F. A. Dunsmoor, J. F. Corbett, E. M. Sala, Van Buren Knott, W. H. Magie, J. R. Eastman, T. C. Witherspoon, E. H. Beckman, M. L. Harris, B. B. Davis, and S. C. Beede.

Dr. John E. Summers, of Omaha, read a paper entitled, "The Differential Diagnosis Between the Acute Pathological Perforations Within the Abdomen, with Special Reference to Duodenal Perforations. The Indicated Technic of the Operation." The paper was discussed by Drs. W. F. Reynolds and A. F. Jonas.

Dr. William Grant, of Denver, read a paper entitled, "The Excessive Mortality of High Intestinal Obstruction." The paper was discussed by Drs. John P. Lord and A. W. Abbott.

Dr. Arthur E. Benjamin, of Minneapolis, read a paper entitled, "Substitute Operations for the More Radical Surgical Work Advised by Mr. Lane in Intestinal Stasis." The paper was discussed by Drs.

J. F. Corbett, C. E. Black, J. R. Eastman, J. N. Jackson, Leonard Freeman, M. L. Harris, and W. T. Reynolds.

President B. B. Davis said: "I wish to apologize for letting these speakers, most of them, speak overtime. They were so eloquent that they literally made time stand still. I think this has been a very profitable discussion."

On motion, Dr. Alonzo M. Pond's paper was postponed until the morning session.

The Association then adjourned until 9:00 A. M. Saturday, December 19.

December 19—Second Day—Morning Session

The Association met at 9:00 A. M. and was called to order by the President.

Dr. Alonzo M. Pond, of Dubuque, Iowa, read a paper entitled, "The Surgical Treatment of Large Inguinal Herniæ." The paper was discussed by Drs. E. P. Quain, of Bismarck, and Dr. Charles D. Lockwood, of Pasadena.

The President, Dr. Davis, announced that Dr. Babler would be allowed to read his paper next, if there was no objection, in order to allow him to catch his train.

Dr. Edmund A. Babler, of St. Louis, then read a paper entitled, "Cavernous and Cystic Lymphangioma of the Cecum." The paper was discussed by Drs. A. E. Hertzler, of Kansas City, and E. H. Beckman, of Rochester.

Dr. Thomas C. Witherspoon read a paper entitled, "Hodgkin's Disease." The paper was discussed by

Drs. E. H. Beckman, of Rochester; Emil Beck, of Chicago, A. F. Evans, of Wichita, and D. P. Mayhew, of Colorado Springs.

The President, Dr. B. B. Davis, said: "I understand that Dr. A. A. Kerr, of Salt Lake City, and Dr. F. G. Connell, of Oshkosh, are neither of them able to be present, but have sent their papers. What shall be done with them?"

On motion, both papers were ordered to be read by title and printed with the transactions.

Dr. Horace G. Wetherill, of Denver: Is this the proper time to present a resolution or report of a committee?

Dr. B. B. Davis, presiding, said: I think it might as well be presented now.

Dr. Horace G. Wetherill: Mr. President, the committee appointed to consider and report upon the President's Address desires to make the following report:

Your committee appointed to consider the address of our President desires to report that we recommend the endorsement by the Association of the suggestions contained in the address, but that we particularly commend for the approval of the Association those which refer to the betterment of surgical practice and surgeons through standardization.

Therefore, we desire to present the following resolution:

Resolved, That the standardization of surgical practice, surgical qualifications, and of surgeons is eminently to be desired, and that the Western Surgical Association approves such means as may be brought to bear for the attainment of this desirable end.

H. G. Wetherill, M. D.
J. R. Eastman, M. D.
E. P. Quain, M. D.

This motion was carried unanimously.

Dr. Van Buren Knott, of Sioux City, then read a

paper entitled, "Carcinoma of the Ovary in an Eleven-Year-Old Girl." The paper was discussed by Drs. A. W. Abbott, of Minneapolis, A. E. Hertzler, of Kansas City, Charles D. Lockwood, of Pasadena, Everett, a visitor, and T. C. Witherspoon, of Butte.

Dr. Willard D. Haines, of Cincinnati, read a paper entitled, "Tumors of the Mediastinum."

Second Day—Afternoon Session

The President, Dr. B. B. Davis, called the Association to order at 2:00 p. m.

The Secretary, Dr. Arthur T. Mann, of Minneapolis, presented the report of the Council.

Report of the Executive Council

There were present at the meetings of the Executive Council, Drs. Jackson, Abbott, Grant, Harris, and Mann.

The following are recommended for membership:

Hare, Earle R.—Minneapolis, Minn.
Salter, P. Harold—Norfolk, Neb.
Richter, Harry M.—Chicago, Ill.
Balfour, Donald C.—Rochester, Minn.
Henderson, James P.—Kansas City, Mo.
Sanders, E. M.—Nashville, Tenn.
Rowan, Charles J.—Iowa City, Ia.
McGraw, Henry R.—Denver, Colo.
Hegner, Casper Frank—Denver, Colo.
Cubbins, Wm. R.—Chicago, Ill.
Martin, Paul F.—Indianapolis, Ind.
Clark, Edmund D.—Indianapolis, Ind.
Tennant, Channery E.—Denver, Colo.

The following are recommended as officers for the coming year:

President—J. Rilus Eastman, Indianapolis.
First Vice-President—Van Buren Knott, Sioux City, Ia.

Second Vice-President—Carl E. Black, Jacksonville, Ill.

Secretary and Treasurer—Arthur T. Mann, Minneapolis, Minn.

For the Executive Council—Drs. W. W. Grant and M. L. Harris.

For the Board of Governors of the American College of Surgeons as the three candidates from this Society—Leonard Freeman, D. S. Fairchild, Sr., and B. B. Davis.

The treasurer's report was approved and is as follows:

The Treasurer's Report

RECEIPTS

Balance on hand Dec. 20, 1913	$287.57	
One volume of the Transactions	2.50	
Fees and dues to Dec. 19, 1914	1,430.00	
Guest tickets to banquet	55.00	
Total		$1,775.07

DISBURSEMENTS

Check returned	$10.00	
Paid for printing	46.25	
Paid for reporting St. Louis meeting	137.62	
Paid for 250 vols. Transactions and cuts	865.88	
Honorarium to Secretary	100.00	
Buttons	26.49	
Postage, express, etc	24.30	
Banquet at Denver	270.00	
Total		$1,480.54
Balance		$294.53

As a place of meeting for next year, Des Moines, Iowa, with Oliver J. Fay, Des Moines, as Chairman of Committee of Arrangements, and the date Friday and Saturday of the week before Christmas.

The report of the Council was adopted by motion.

Dr. Arthur T. Mann: The chairman of the Committee of Arrangements suggests that a resolution of

thanks be sent to management of the Hot Springs Hotel, Idaho Springs, Colo., which expected to entertain, at its own expense, about fifty surgeons. None of us have gone, nor do we expect to go, as I understand. I should like to propose that we send such a resolution.

This was carried without dissent.

The President-elect, Dr. Joseph R. Eastman, of Indianapolis, was conducted to the chair by a committee, consisting of Drs. Perry and Littig, appointed by President Davis. Dr. Eastman said:

There is an old joke about a medical student who was asked by his teacher of surgery what he would do first thing if he were called to see a man who had been blown up into the air two or three thousand feet by dynamite; and the student said, Well, first of all, I believe I would wait until he came down. (Laughter.)

I feel decidedly up in the air at this honor conferred upon me, so that I can hardly find words to express my thoughts.

I hope you will acquit me of affectation when I say that I feel very deeply that a very much worthier man could have been found for this honor than I. But I appreciate it from the bottom of my heart and I will do my very best to conduct myself in such a manner as to reflect as little disgrace as possible upon the Western Surgical Association.

President Davis resumed the chair.

Dr. James F. Percy, of Galesburg, Ill., read a paper entitled "Report of the Chairman of the First General Study Tour of the Western Surgical Association."

President Davis: I am sure the Association appreciates this paper and this report. Is it desired to have any discussion on the paper? Or to make any special orders in reference to the paper?

Dr. J. N. Jackson: I should like to make simply one suggestion at this time: Those who participated in that trip had a most delightful and most profitable experience; and, as I believe, the idea of taking a trip is a most excellent one. I am sure those who had this experience are convinced that that is so.

As it takes time for the preparation of these trips I would suggest at this time, and I move, that a committee be appointed by our President to consider the advisability of a trip to be taken in 1916, possibly to Hawaii, the Philippine Islands and Japan.

This motion was seconded and carried unanimously.

Dr. John P. Lord: I think it is only proper for us to express in a resolution our appreciation of this report made by Dr. Percy.

It necessarily has meant a great deal of painstaking care exercised throughout the whole trip, and it has, of course, required some labor to prepare his paper.

We all appreciate it most highly, and I therefore move you that it be the sense of this Association that we do appreciate and thank Dr. Percy for his handsome efforts.

This motion was seconded and carried unanimously.

Dr. Horace G. Wetherill, of Denver, read a paper entitled "A Plea for Higher Hospital Efficiency and Standardization." This paper was discussed by Drs. E. P. Quain, of Bismarck, Lawrence W. Littig, of Davenport, D. S. Fairchild, Sr., of Clinton, M. L. Harris, of Chicago, C. E. Black, of

Jacksonville, J. N. Jackson, of Kansas City, and President B. B. Davis, of Omaha.

Dr. John P. Lord: I move that the President-elect appoint a committee of three to take such measures as are considered best in presenting this matter formally to the Carnegie Foundation.

This motion was duly seconded.

President Davis: It is moved and seconded that the President-elect be authorized to appoint a committee of three for the purpose of getting this before the Carnegie Foundation.

Are there any remarks?

Dr. M. L. Harris: We all know that the American Medical Association has taken up this matter, and that a committee is already doing very effective work. I think if the matter is to be brought to the Carnegie Foundation it should be done with all of the authority that we can possibly get behind it, and all the men we can get behind it. For this Association alone to bring it to the Foundation without the coöperation and support of these other organizations I think would be a mistake.

I would rather see this Association appoint a committee to coöperate with the American Medical Association and other associations, to see if we cannot round up the entire profession in a movement to bring it before this body, rather than have this Association alone undertake it. Therefore I would like to amend this motion to the effect that this committee take up the matter, and work in conjunction with the committee of the American Medical Association, to see if some movement cannot be brought about to bring it before this Foundation, rather than have this committee alone attempt the work.

Dr. Lord: I heartily agree with Dr. Harris' suggestion, and I gladly accept the amendment, or to have it go as a substitute motion. I think we should work in coöperation.

President Davis: Then this committee will be appointed to coöperate with the American Medical Association and other organizations.

Dr. H. G. Wetherill: May I say that the American College of Surgeons, the Philadelphia County Medical Society, and one other important association have already taken such action, to bring this matter to the Carnegie Foundation through committees. There are at least four that I know of.

The question was here called for, and on being put was carried unanimously.

President Davis: I would suggest the President-elect need not necessarily appoint that committee this afternoon, but take time to select such committee as he feels will do the work.

Dr. Oliver J. Fay, of Des Moines, read a paper entitled "Anaphylactic Reaction Following Operation for Echinococcus Cyst."

Dr. J. Frank Corbett, of Minneapolis, read a paper entitled "The Relation of Ductless Glands to Shock; Experimental Work." The paper was discussed by Drs. J. P. Aylen, of Fargo, Arthur E. Hertzler, of Kansas City, and A. W. Abbott, of Minneapolis.

President Davis: The traveling committee to be appointed for the purpose of arranging the trip to the Orient, which I felt under the circumstances the President-elect should appoint, he has appointed and asked me to announce, as follows:

Dr. J. F. Percy, Galesburg, Ill.
Dr. Leonard Freeman, Denver, Colo.
Dr. Jabez N. Jackson, Kansas City, Mo.
They will constitute the committee.

Dr. Arthur E. Hertzler, of Kansas City, from notes, gave a paper entitled "Local Anesthesia in the Prevention of After-Pain and Shock." This paper was discussed by Drs. M. L. Harris, of Chicago, A. A. Law, of Minneapolis, J. F. Corbett, of Minneapolis, Donald Macrae, of Council Bluffs, W. W. Grant, of Denver, and F. A. Dunsmoor, of Minneapolis.

The paper by Dr. John N. Warren, of Sioux City, entitled "A Study of the Sacro-iliac Articulation," was read by title in the absence of Dr. Warren, who was not able to be present.

Dr. Hertzler found it necessary to leave before the conclusion of the discussion on his paper in order to catch his train; and Dr. M. L. Harris, of Chicago, was asked to close the discussion for him at this time.

It was moved and seconded, and carried unanimously, that those of the local profession be given a vote of thanks for all which has been done for the visiting members of the Western Surgical Association.

President's Address

TEAM-WORK

Byron B. Davis, M. D., F. A. C. S.

OMAHA, NEBRASKA

In the modern evolution of surgery conditions have so changed that the methods of work practiced a few years ago must be modified to fit in with recent developments. It is now found impossible for a man in any branch of the profession to work alone as he once did. So much more can be accomplished when several men join forces that the solitary worker is likely to be lost or at least fail to score.

The surgeon ought to know a great deal about anatomy, physiology, chemistry, pathology, bacteriology, physical diagnosis, röntgenology, and be an accomplished physician and a man of almost superhuman judgment. But the man who only expects to live seventy years cannot hope to attain commanding efficiency in all these branches and he finds it necessary to call to his assistance those who are experts in their special fields.

A simple case often needs contributions from practically every department of medical knowledge before a thoroughly comprehensive diagnosis can be worked out. Suppose that Dr. A. prefers to work alone. He examines his case physically in such a way as to include every organ; makes his own blood-examina-

tion, chemical analyses of the gastric contents, feces, and urine; bacterial analyses of the several secretions, a neurological examination, an *x*-ray and ophthalmological examination; does the required operation himself and makes the needful pathological examination of the tissue removed. If he can do all these things and do them well, I take off my hat to him; but, I question his wisdom.

Working in such a way he could not adequately dispose of more than two or three cases a week, and, if a case were urgent, might fail to reach a definite conclusion until after the patient had ceased to need his services. His experience in each of the expert steps would be so limited that he could not attain the efficiency of the man who contents himself to cover a narrower field.

The man who makes twenty blood-counts to his one, or twenty urinary or gastric analyses, or examines twenty hearts and lungs, or inspects the fundus of twenty eyes, or makes and interprets twenty *x*-ray plates, or studies the bacteria of twenty cases, or studies the gross and microscopial pathology of twenty specimens, or performs twenty similar surgical operations, is bound to be more efficient. The special worker's judgment in his own work cannot fail to be more valuable than the judgment of the man who tries to cover the entire field.

Dr. B. is much wiser and more efficient by perfecting himself in one line and getting his information with reference to the many special tests from those who have had expert training and wide experience in their respective fields. When he brings all his facts together from these several sources he is less liable to err in his diagnosis than the man who works alone.

This is what I understand by "team-work." Each individual member of the team assists, and they correct and check up one another.

The chief danger in team-work is in not taking a comprehensive view of the case as a whole after all the isolated facts have been brought together. No surgeon can safely make a final diagnosis in a complicated case until he has assimilated all the findings, and subjected them to a rigid and logical analysis. Good team-work will permit fewer surprises at the operating-table, the mortality from operations will be lowered, and the results, immediate and remote, will be more satisfactory.

STANDARDIZATION OF THE SURGEON

What are the fundamental qualities necessary in a man without which he cannot become a surgeon?

The young man with surgical ambitions should be clear-headed and logical. His mind should be able to think straight, and grasp a subject comprehensively, should be quick in making a decision, able to weigh all the factors in a proposition, distinguishing between essentials and non-essentials, but giving each item its proper perspective. He must have courage of a high order. The timid mind and the faint heart are poor props to lean on when life and death are at stake. He must have physical and mental energy, have a sound mind free from morbidness,—a mind that can be depended on under the most trying circumstances. He must be capable of marshalling all his knowledge on a given subject quickly when it is needed. The slow thinker loses time when time may mean life or death. The slovenly thinker is likely to have essential facts come to him after the operation is over.

One of the prerequisite qualities is sanity. The safe surgeon's wits are never wool-gathering, but go straight to the point by the most direct route.

The ability to operate skillfully is on a lower level than skill in diagnosis, ability to size up a case in all its bearings, an almost uncanny comprehension of a patient's resisting power; but no surgeon can attain marked success who has not skill with his hands. The manual bungler should never try to be a surgeon.

The would-be surgeon should be a man who loves life, and has the highest regard for the life of his fellow. He must be willing to do his utmost for every case entrusted to his care. He must be warm-hearted, have wide sympathy, and be so devoted to his profession that the very idea of graft is repugnant to him.

Clean personal habits, both moral and physical, are not too much to demand. An agreeable personality that begets confidence, combined with a cheery hopeful disposition and backed up by a dogged persistency and a spirit never to give up, will be most valuable assets.

Having selected a man whose personal qualities seem fitted to make the raw material for a future surgeon, what training is necessary to insure the best finished product? what minimum requirements should be exacted before a man is permitted to go out and practice surgery? Is the present system, which regards the mere possession of an omnibus degree in medicine, surgery, and the allied branches, a proof of fitness to practice surgery? Is it fair to the public?

Of late a great deal has been said for and against the standardization of the surgeon. There is a better way to develop a surgeon than to follow the old law of "the survival of the fittest." The unfit usually decide finally that they are unfit, but at what a fearful cost! Those who finally succeed, and are classed with the "fit," too often reach this goal by a route that has to be paid for by the innocent public.

It seems more humane, more decent, and more in

keeping with the high aim of surgery, that a course of study and work be devised which will fit men for the practice of this high vocation; and that those unwilling, or unfitted, to follow the prescribed course, be denied the right to practice major surgery. With such a system in operation, the layman who entrusts his life or the life of his child to a surgeon, will have assurance that the man selected has reasonable qualifications and training for his work.

The working out of the details of a special preparation for the surgeon will be an arduous task, calling for the best wisdom of our wisest men; but it must be done, or our state legislatures will do it for us. Those long engaged in the practice of surgery, who have attained undoubted success and who have lofty ideals, are so much better qualified to work out the problem that it behooves them to shoulder the burden.

It is not my purpose to do more than make suggestions along general lines. There can be no difference in opinion about the wisdom of the candidate for surgery first being grounded in the fundamentals by completing the work that leads to the degree of doctor in medicine conferred by chartered colleges. The wider the culture preceding the beginning of the work of the medical college, the better the average results.

As a next step following the M. D. degree I believe an interneship in a hospital where thorough scientific and systematic work is done, is of the utmost importance. After this the most practical training would seem to be a term of years as apprentice and assistant to a surgeon of skill and ability. To share daily with the busy surgeon in working out the cases, developing the diagnosis, deciding which should be operated on and which should be treated by other methods, sizing up the operative risks, assisting at the operations, and sharing in the after-treatment, will bring out and

develop a young man if he has in him the stuff of which surgeons are made.

After a time the apprentice may be entrusted with minor operations under the eye and guidance of his chief. Little by little it will be safe to drill him in the performance of operations of greater magnitude until, imperceptibly, he comes into his own with a technic and a matured judgment and without risking lives unnecessarily during the process.

It might be found advantageous for the assistants not to remain with one surgeon during the length of their apprenticeship, but to change from one to another, in order to gain the benefit of a varied technic and different methods of study of cases and treatment.

The process for finally passing on the fitness of a man to enter into practice as an independent surgeon, is another problem to be solved. Shall it consist of a special degree by an authorized university or by special license by the state? Shall it emanate from a national board to be established by the general government, or shall the American College of Surgeons be made by general consent the licensing body? It has seemed to me entirely safe to recognize membership in the College of Surgeons as a badge carrying with it the right to practice surgery.

The manner in which the examination is conducted, is of great importance. The test to ascertain the candidate's knowledge is easy. To ascertain whether he is a real surgeon, safe and sane, possessed of the requisite technical skill, endowed with the sterling mental and moral qualities which are so important, is a more difficult matter. A term of service with a number of different surgeons of standing, who shall act as a committee to pass on these points, ought to be fair, and would minimize the chances for mistakes.

In connection with this subject let it be distinctly understood that the right to do any operation of emergency, or to do the usual routine surgery that comes to the hand of any physician, is unquestioned. Only the setting one's self up as an expert in surgery is under discussion. The proposals do not seem to me to work any hardships on anyone honestly and conscientiously desiring to fit himself as a surgeon. The chief object of the proposed arrangement is to do away with some grave abuses which we all know exist; and such an arrangement will enable the young man of ability to take a high place in the ranks in less time, and by an infinitely more satisfactory road, than under the present catch-as-catch-can process.

THE CANCER QUESTION

The gravest question confronting the medical profession today is how best to deal with cancer. So many of the deaths from this disease might be prevented that it seems criminal to fail longer in carrying out an educational campaign which has for its purpose bringing hope to the victim of cancer.

The task will prove a difficult one, and will require zeal to carry it on and patience to wait for results. There seems such a widespread feeling that the diagnosis of cancer is equivalent to having the death sentence pronounced, that it will require persistent, earnest effort to allay the prejudice against operation.

In the past quarter century while the mortality from tuberculosis, typhoid fever, cholera, yellow fever, smallpox, appendicitis, gall-bladder disease, diphtheria, gastro-intestinal diseases of children, etc., has been steadily decreasing, the mortality from cancer has been increasing. To show that this increase is real, and not fanciful, let me quote from the statistics of the state of New York:

1887 to 1889.....43.0 deaths per 100,000 population
1890 to 1892.....48.2 deaths per 100,000 population
1893 to 1895.....50.2 deaths per 100,000 population
1896 to 1898.....58.7 deaths per 100,000 population
1899 to 1901.....66.0 deaths per 100,000 population
1902 to 1904.....69.4 deaths per 100,000 population
1905 to 1907.....75.1 deaths per 100,000 population
1908 to 1910.....79.1 deaths per 100,000 population
1911 to 1913.....86.5 deaths per 100,000 population

Thus, in the twenty-seven years from 1887 to 1913, inclusive, the death-rate from cancer has more than doubled. This is about the rate of increase for the United States as a whole.

An explanation, in part, of this apparently alarming increase is that diagnosis is much more positive now than a quarter of a century ago. According to some recently published records of findings in the Berlin hospitals, twenty per cent of the deaths from cancer found at autopsies are unrecognized clinically.

It is proper to concede that a part of the apparent increase in cancer is due to better diagnosis; but I believe that this is only one-half the explanation, and that there is a real increase. I do not mean to assert that the tendency for cancer to develop is greater than it was twenty-five years ago, but that the many life-saving discoveries and sanitary improvements are carrying over a large and increasingly larger number of individuals to the cancer age.

If it were possible to compare the number of deaths from cancer per annum in each 100,000 of the population above forty years of age during the last quarter century the rate of increase would be much less,—only such an increase as might easily be accounted for by improved diagnostic methods. I do not believe that an individual between the ages, say of forty and sixty years, is in any more danger of falling a victim to

cancer than was an individual of the same age twenty-five years ago.

If this reasoning is correct every successful effort to save the lives of the young and middle-aged, and to carry them over to the more advanced age, only adds to the number of cancer victims. Cancer stands like a beast of prey, gloating over modern life-saving devices, like the safety coupler, diphtheria antitoxin, vaccination, the prevention of yellow fever, cholera, tuberculosis, and other contagious diseases, the modern treatment of appendicitis, the use of clean milk, and better infant hygiene, because all these help to swell the army of his victims!

The world has waited long enough for the discovery of a wonderful prophylactic or curative serum which will remove the cancer terror by one fell swoop. It will be necessary to make the best of the present knowledge; and especially a more careful and systematic study of the early symptoms should be made.

One of the great mistakes of the past has been in withholding from the general public a better understanding of the early signs of cancer, and the fact that, when discovered early, it is, in a large proportion of cases, curable.

An organized campaign to show the people that early radical operation will save the great majority of cancer sufferers, is the crying need of the times. The public will have to learn that the mortality from cancer is directly proportional to the length of time that elapses between the date of origin of the disease and the date of its radical removal; that cancer is even more an emergency disease than appendicitis.

The almost universal feeling among laymen that when cancer is present the case is hopeless, will have to give way to the sentiment that when cancer develops the time for fighting has come. Until this change

in the mental attitude is reached there can be little gain over the present results.

At present our mortality statistics of cancer have been based on cases relatively advanced. Many cases are undertaken that the surgeon realizes at the time are well-nigh hopeless. As the records now run in most hospitals, about twelve to thirteen months on an average elapse between the time of the discovery of the suspicious lump or symptom and the date of the operation. This is about as sweeping a denunciation of the prevalent method of dealing with a terrible malady as could be made. This means there are now in the United States more than one hundred thousand persons suffering from cancers that should be operated on, and that will be operated on within the next twelve months. Their chances of permanent cure are decreasing every day.

As a result of the large proportion of late operations, the operative mortality is unnecessarily high, and the number of recurrences so great that layman and doctor alike are disheartened and inclined to ask, What is the use?

In recording cases it seems only fair to draw a sharp distinction between the early and the advanced ones. This should be done at the time of the operation. The cases should then be followed, and in a few years the contrast between the results of the early and the late operation will be so striking that it cannot fail to convince the public, and will go far toward bolstering up the courage of our rather timid profession.

It may seem like the pratings of an idealist, but I am convinced that the time is coming—and I expect to live to see it—when cancer will largely lose its terrors, when the victim of the disease will report promptly to his doctor the least sign that might be indicative of incipient cancer, and when the doctor,

thus consulted, will not dismiss the case with a shrug and the advice to wait and see what develops, but will investigate the case to its utmost limits. It will then no longer be customary to consider a growth benign until it proves itself malignant, but the viewpoint will be reversed. More operations on suspicion may be done, but myriads of lives will be saved that are now sacrificed as an offering to the present watchless waiting policy.

Missionary work of a very comprehensive character will be required to bring about such a change in sentiment and practice. Wise publicity seems to be the only remedy.

Such an educational campaign needs to be carried on as will reach all classes and conditions. Not emotionalism, not an alarmist propaganda, but clear, concise, logical statements of facts will be most likely to get the people's attention and convince them.

Efforts at publicity cannot succeed if spasmodic, but there should be a steady, persistent presentation of the proven facts in a simple way. People need to be convinced that cancer is curable, and taught as much as possible of the early manifestations of the disease in its more usual haunts.

It is rather apparent that members of the medical profession need enlightenment also. They need to have brought home to them their serious responsibility when consulted in reference to any condition in the least suggestive of malignancy. A little bolstering up all along the line would not be wasted effort. Every physician needs to have clearly brought home to him the difference in prognosis when the operation is done while the cancer is early and still local, or delayed until it is neither early nor local.

Another item of importance is a more careful study and enlightenment on the so-called precancerous con-

ditions and the lesions and conditions that seem to serve as causative factors. Irritable scars, imperfectly healed ulcers, warts, moles, fissures, and the like, form the starting-point for cancer so often that they cannot consistently be ignored.

The greatest forward step ever made looking to lowering the mortality was the organization of the American Society for the Control of Cancer. This society was formed in the spring of 1913, and has already done much good. Its fundamental object is to investigate and reduce to concrete form all the known facts about cancer, and to serve as a center for enlightening the lay and medical public of all the essential facts.

One year ago at the meeting in St. Louis the Western Surgical Association adopted a resolution endorsing this society, and pledging its support. With the backing of the best men in the profession, and with the powerful aid of many benevolent citizens who have become interested, there is little doubt that within a short time the splendid results achieved by the National Association for the Prevention and Study of Tuberculosis will be duplicated, and cancer will no longer be a household dread.

REPORT OF CASE OF POSTOPERATIVE ANURIA OCCURRING THE TWELFTH DAY FOLLOWING NEPHRECTOMY FOR HYPERNEPHROMA

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That anuria may follow certain surgical operations is a well-known fact. The gravity of the operation appears to be of less importance as a determining factor in the causation of suppression of the urine than the structures concerned in the operation. For example, complete suppression of the urine has so often followed the use of the catheter for the relief of a greatly distended bladder that surgeons of former times were wont to caution their pupils against the practice of completely evacuating the bladder at the first sitting.

Unskillful and maladroit instrumentation is more likely to be followed by suppression of the urine than when the manipulations are carried out with true surgical skill. Even in some of the cases of major operations where convalescence has been interrupted by the occurrence of anuria, it has sometimes been thought that the way in which the tissues were manipulated during the course of the operation may have been partly responsible for the unfortunate complication. For example, when nephrectomy, or some other operation upon the kidney, has been followed by complete cessation of the renal functionment the occurrence has

been attributed to traumatism of the healthy organ by the roll or sandbag used on the operating-table pressing the kidney against the vertebral column. This looks like an improbable accident, but there may be some truth in it.

Any operation followed by shock may cause an oliguria or even anuria. The operations, however, which are oftenest complicated by postoperative urinnary suppression are operations upon the urogenital system. The ligature method for the cure of hemorrhoids, and the operation of appendectomy, are among the procedures which have sometimes given rise to anuria. In some instances an unrecognized acute nephritis may have played an important rôle in the case. When the anuria is due to surgical shock it usually follows the operation immediately. In fact, a marked oliguria is not an infrequent sequel during the first twenty-four or forty-eight hours after a major pelvic operation.

The frequency of this postoperative phenomenon has been remarkably diminished since the almost universal practice of hypodermo- and proctoclysis with normal salines has been the vogue.

The case of postoperative anuria which constitutes the subject of this communication, as it appears to me, is fraught with much more than ordinary interest. The cessation of the urinary function did not take place immediately after the operation, but was delayed almost a fortnight, and continued unmitigated for a like period of time.

Case.—Mrs. G., aged 28, came under my observation on July 25, 1913. Married, mother of two children. Both parents and both maternal grand-parents are still living and in good health. The grand-father is 87, and the grand-mother is about 80 years old. No very definite information could be elicited concerning the

grand-parents on the paternal side, save that they both died at a very advanced age.

The patient has two brothers and two sisters, all living and in good health. She menstruated first at the age of twelve. Her periods were always regular, and free from pain, until about three months prior to coming under my observation, when she began to suffer greatly at each menstrual epoch.

The earliest clinical manifestation which can be associated with the immediate history of her disease was a severe and persistent pain in the back, which was first observed in September, 1912. For this she sought the advice of her family physician, who is a very excellent medical man, and who made a diagnosis of lumbago, and treated the case without success. She continued to suffer, and in April, 1913, developed an infection of the upper part of the left popliteal region, which terminated in an abscess that was treated by her regular medical attendant with cataplasmata and incision with success. A month later, in May, 1913, she began to suffer severely with pain situated in the region of the right kidney with tenderness in front and medianward. This attack continued unabated for a week. About four weeks later there was a recurrence of the painful condition, which lasted nearly a month. After a respite of two weeks there was another recurrence. Each succeeding attack was described as having been more severe than the one preceding.

By this time, when the patient came under my observation, the kidney could be palpated as a very sensitive mass, indeed, in the right upper quadrant of the abdomen. The mass extended well toward the median line. There were almost no abnormal urinary findings, notwithstanding that by bimanual palpation with the finger-tips of the left hand in the costo-ver-

tebral angle of the right twelfth rib, and those of the right hand over the anterior and inner aspect of the tumor mass it could be demonstrated that the neoplasm was in all probability continuous with the kidney. It was further observed that the ankles and knees were swollen and painful; and in a lesser degree the wrists and elbow-joints were likewise affected. This condition had prevailed for a month. Gradually the swelling had extended from the ankles up to the knees, and the patient was now unable to walk. There was a bright-red maculoid eruption which faded away into brownish blotches covering the inferior extremities, and which resembled Henoch's purpura. I do not regard the articular nor the cutaneous manifestations as bearing any important relation to the renal neoplasm nor the anuria that followed its removal, but they coëxisted and therefore must receive mention.

The patient had become emaciated, and much reduced in strength, as a result of pain and lack of ability to nourish herself. She was admitted to St. Francis Hospital on July 27, 1913. Matinal temperature, 99.8°; vesper temperature, 100°. Pulse, 90; leucocytes 8,000. The urine was alkaline and free from albumin, sugar, casts, pus, bile, bacteria, and blood. The quantity of urine was near normal. The *x*-ray failed to give any definite information further than to show a large shadow of the kidney.

Gall-bladder trouble was excluded because of the absence of the usual syndrome accompanying disease of the biliary passages. A neoplasm of the cecum or ascending colon was likewise excluded because of too little disturbance in the intestinal functions, absence of blood in the alvine dejecta, and signs of obstruction. Stone in the kidney was excluded on account of negative urinary findings and failure of the *x*-ray pictures to disclose anything. Tuberculosis of the

kidney was considered, but excluded for lack of positive urinary findings, and it was thought that the elevation of temperature and perhaps the acceleration of the pulse were attributable to the articular manifestations.

With the tentative diagnosis of renal neoplasm in mind, the patient was prepared for operation by the administration of aperient agents, a dietary of liquids, including plenty of water, cereals, and vegetables, and baths to place the cutaneous system in a favorable condition.

Operation August 2, 1913: Under ether-oxygen anesthesia an exploratory incision was made over the left kidney for the purpose of palpating and inspecting that organ as to its presence, or absence, size, and apparent capacity to perform its own work and that of its fellow. The left kidney being apparently normal, the exploratory wound was closed. The patient was now placed in the position for right lumbar nephrectomy. The operation was accomplished with some difficulty on account of the density of the adhesions encountered. Fortunately the strongest adhesions were between the posterior surface of the kidney and the lumbar structures, the tissues being fused so as to obliterate the different layers. The tumor had apparently propagated itself through the capsule, and encroached upon the adjacent structures in the loin. It was necessary to remove considerable muscle and fascia in order to enhance the patient's chances for prolonged or permanent immunity. For the same reason the pedicle was made as short as consistent with safety. The neoplasm was situated in the cortical portion of the kidney, which may account for the absence of abnormal urinary findings. The specimen was sent to the laboratory of the hospital, and was reported upon as a malignant hypernephroma.

The patient came out of the operation in very good

condition, and her convalescence was satisfactory until the twelfth day. The urine was voided several times a day, and the quantity was sufficient.

On August 14, twelve days after the operation, she had a molimen menstruale attended with considerable pain and only a scanty flow. The renal function was suddenly suspended, no urine whatsoever being voided. The next day, August 15, after the administration of café noir per rectum the nurse was able to obtain almost half an ounce of dreggy fluid from the bladder with the aid of the catheter. Later in the day the catheter was employed again, and but a single drachm of dirty-looking fluid was brought away. One-sixth grain of pilocarpine hydrochlorate was administered hypodermically. This was soon followed by an almost incredibly profuse diaphoresis, which was always copious.

The wound now being healed, the patient was placed in the hot bath once a day as an additional means of stimulating still further cutaneous transpiration. When the patient was removed from the hot bath, placed under the blankets, and given her hypodermic dose of the pilocarpine salt, prompt and profuse sudation always followed. The patient was nourished exclusively with fluids. There were no marked uremic manifestations exhibited at any time, notwithstanding the facial expression depicted a certain amount of anxiety after the first three or four days. Hysterical phenomena were not to be observed at any time. On August 24 a few drachms of dark muddy albuminous urine were voided. On August 25, eleven days after the onset of the anuria, seventy-five ounces of urine were voided. The daily quantity of urine from this date on remained between forty and fifty ounces, and her further convalescence was uninterrupted.

She was seen a few days ago, sixteen months after her operation, and was still feeling well.

DISCUSSION

Dr. Charles D. Lockwood (Pasadena, Cal.): It seems to me this paper ought not to be passed over without comment on the fact that no valuable information was obtained as to the opposite kidney before the operation was performed. The time has gone by when we should rely on an exploratory incision to determine the condition of the opposite kidney, because we know but little more then than the presence of this kidney.

Moreover, the report does not show the total quantity of urine nor urea, nor specific gravity, which, I believe, are the three most important factors in determining the functioning of the kidney, and advisability of operation in these cases.

I think it quite possible if the functional test had been applied to this kidney, that is, the supposedly good kidney, it might have shown conditions that would have led to preliminary treatment that might have avoided subsequent anuria.

Dr. James F. Percy (Galesburg, Ill.): I want to call the attention of the Association to a paper* which I read last year before the A. M. A., in which I suggested the use of thyroid extract in this class of cases. I am positive if this drug had been used in this case within twenty-four hours, the anuria would have disappeared. My dosage would be four grains three times a day, to begin with, and increased six grains each day until the kidneys resumed their function or the patient showed symptoms of hyperthyroidism.

Dr. J. N. Jackson (Kansas City, Mo.): I want merely to mention a case which came under my observation recently which is quite apropos to the report of the case of Dr. Basham, and likewise has some pertinence to the remarks of Dr. Lockwood.

A patient came under my observation with a history of having had trouble about eighteen years before. Fourteen years before she came into my hands she was operated on by another doctor, at which time, as I understood it, the operation was done for a swelling.

After this operation she continued to have pyuria, which caused a great deal of trouble. About six months before

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*Nephritis: Its Treatment with Thyroid as a Preliminary to Operation, *Journal of the American Medical Association,* August 9, 1913, vol. lxi, pp. 380-383.

coming under my observation she developed a swelling in the region of the left kidney, caused by a tumor. When this was opened there were left two or three persistent sinuses. She was brought to me on account of the persistency of these sinuses, although the woman looked to be unusually healthy, physically well developed, and all that.

A cystoscopic examination was made, and a functional test made from the urine of the right kidney, which apparently was normal. The secretion was apparently normal, and we had reason to assume from the examination that the right kidney was in good condition. We operated and removed, therefore, the left kidney, the upper half of which proved to be two or three times its normal size, containing tubercular pus. The operation was very difficult on account of the adhesions. Notwithstanding this she had no trouble whatsoever so far as shock was concerned.

The interesting thing with reference to the subject of Dr. Basham is, that that woman went for eleven days after the operation, not passing a dram of urine in the entire time, and yet not showing the slightest physical signs of any disturbance. As a matter of fact, on the tenth day she asked if she could not go home, stating she did not see any reason why she should stay in the hospital, as she was feeling well. She was sitting up.

The efforts to secure a flow of urine had been absolutely without avail. On the thirteenth day after the operation she first began to show some signs in the facial muscles, and from then she developed uremia, and in about three days died.

In this case the autopsy showed that the right kidney was the worse of the two, about one-half of the left kidney, which had been removed, being in good condition.

This case is interesting as showing two or three things: first, the long duration of complete anuria without any symptoms apparently of uremia; and, second, despite the catheterization of the ureter, and the functional tests of the kidney, that the right kidney, which was shot all to pieces, showed nothing on examination.

Dr. Basham (closing): I have nothing further to add. An experience similar to that related by Dr. Jackson has caused me to take the method of exploration of the kidney instead of trusting to the ureteral catheter. Besides, an exploratory operation is not as harmful to a patient in serious condition as ureteral catheterization. And besides all that,

you may have a horseshoe kidney to deal with, or, you may have a kidney that has two ureters. A good many abnormal conditions may prevail which may mislead one in any investigation with the catheter.

It is preferable by far to make a simple incision, and examine the kidney, and with experience one can usually tell by the fingers whether the kidney is healthy or not; and you can always tell whether it is there or not.

COMPRESSION OF BOTH COMMON CAROTIDS IN GENERAL IDIOPATHIC EPILEPSY

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Ligation of the large afferent vessels of the brain for the relief of epilepsy is not a new procedure. Although such operations have never received anything approaching general recognition, one may find, in a search of the literature embracing the last few decades, record of many instances of ligation of one or more of the large arteries of the neck in epileptics.

Baracz *(Wien. Med. Woch.,* 1889), Sommer (*Virchow's Arch.,* 1890, cxix, Heft 2, p. 362), Binswanger *(Die Epilepsie),* Wyeth *(Essays on Surgical Anatomy),* Hasse *(Krankheiten des Nerven-systems,* p. 297), and Alexander *(Gowers Dis. of Nerv. Sys.,* vol. 2) have mentioned the favorable effect upon general epilepsy which may be induced by ligation of the carotids or vertebrals. Many years ago Wyeth (op. cit.) observed that "since dilatation of the arterioles and capillaries of the medulla oblongata is accepted by Schroeder, van der Kolk, Niemeyer, and others, as the most constant lesion in epilepsy, I would suggest, and would perform if the opportunity presents, deligation of both vertebral arteries. This would arrest the direct and probably irritating flow of the blood through this ganglion, leaving the recurrent flow from the carotids through the posterior communicating

arteries to supply the necessary amount of nutrition to this portion of the encephalon."

Alexander of Liverpool ligated the vertebral arteries in epilepsy with what seemed at first to be excellent effect, but later reported this method to be without value (Binswanger and Momburg). The investigations of Ritters *(Verh. d deutsch. Ges. f. Chir.,* 1913) sought to explain the failure of vertebral ligation. They apparently made clear that the vertebral arteries play an unimportant rôle in the blood supply of the brain. The carotids, according to Ritters, conduct almost all of the blood to the brain.

Delegeniere *(Arch. Prov. de Chir.* No. 10, 1914) observed excellent results in a case of epilepsy following ligation of the superior longitudinal sinus just above the torcular Herophili. During a trephining, it became necessary to ligate the longitudinal sinus to prevent hemorrhage. Delegeniere regards epilepsy as a clinical syndrome common to many pathologic conditions, and holds that the many causative lesions of epilepsy produce in different ways a permanent dilatation of the veins upon the surface of the cerebral hemispheres. He therefore regards it a logical step to modify the venous circulation of the cortex by ligation of the terminal portion of the longitudinal sinus in the belief that distention of the superficial cerebral veins is the cause of irritation of the cortex. He regards this operation as analogous in its purpose and mode of action to ligation of the upper part of the internal saphenous vein for varicosities of the veins of the leg,—that is, placing a ligature around the longitudinal sinus would, he says, bring about a collapse of the over-distended veins which supply the sinus.

Jonnesco, von Jacksch, Spratling and Park, Joseph Beck, and a number of others, have performed cervical sympathectomy in general epilepsy with a view to

cutting off sensory stimulation from the viscera, and especially to influence directly the circulation of the brain by changes in the caliber of its blood-vessels through the action of the vascular nerves.

Lafforgue (quoted by Spratling and Park, *Jour. of Nerv. and Ment. Dis.,* April, 1905) observed in rabbits, as a direct result of stimulation of the cervical sympathetics, spasmodic ischemia of the whole cerebrum, cerebellum, and medulla oblongata.

Jonnesco concluded that the cervical sympathetics contained vasoconstrictor fibers for the head, face, and heart. This was made evident by electric excitation. What is more important in this connection, Jonnesco further noted that the cervical sympathetics contained vasodilators for the inner part of the cheeks, gums, and lips, and for the brain. This was shown by the application of feeble electric currents.

Winters' table shows that of 122 cases of general epilepsy, treated by cervical sympathectomy, 6.6 per cent were cured, 13.9 per cent temporarily cured, 18.9 per cent improved, 54.9 per cent not improved, and 5.7 per cent died.

The pathogenesis of epilepsy is obscure, and we are still ignorant of the cause of the attack. Many writers consider it unlikely that a palpable anatomic alteration exists. Orloff *(Archiv. für Pschiatrie and Nerven Krankheiten,* vol. 38, Heft 2) found no characteristic changes except thickening of the glia, especially in Ammon's horn, possibly a congenital sclerosis.

The claim of Chaslin, that a gliosis is to be regarded as the cause of epilepsy, is, as stated by Hirt, by no means proven. Much more plausible is the theory that the amount of blood in the brain is of some importance in this connection.

Turner *(Jour. of Mental Science,* vol. liii, No. 220) says that the exciting cause of epileptic attacks is a

sudden stasis in the blood-stream, usually in a limited portion of the cortex.

Bechterew believes that there is an increased blood flow to the brain, and a temporary dilatation of the capillaries during the attack. Bechterew's views were based upon the experiments of Todorsky (Hirt, *Dis. of Gen. Nerv. Sys.*, p. 586). On the other hand, von Leyden and Sommer noted evidence that the exciting cause of the epileptic attack is anemia of the brain. The former observed epileptic attacks in cases of aortic stenosis undoubtedly as the result of a temporarily insufficient blood supply, and the latter remarked their occurrence in a case of ankylosis of the atlas, which had produced narrowing of the vertebral canal in its upper portion.

It has been observed that sodium santoninate injected into rabbits quickens the pulse and respiration. The rabbits' ears become redder and hotter, due to the increased blood supply. This is followed by fine fibrillary contractions. With larger doses, convulsions appear with clonic spasmodic movements, followed by return to the normal. Jelliffe (*Jour. of Mental and Nerv. Dis.*, 1909, p. 252) found in rabbits thus injected with santonin intense congestion of the meninges and of all the cerebral vessels, large and small.

Permanent narrowing of both common carotids was adopted by Momburg for the relief of epilepsy in two cases (*Deutsche Med. Woch.*, April, 1914). Coming accidentally upon an epileptic during a seizure in the street, it occurred to him to apply digital compression to both carotids to determine the effect of such compression upon the typical clonic contractions which were present. With thumb and forefinger he compressed both common carotids, and almost immediately the convulsive movements ceased, the man drew a deep breath, opened his eyes, looked about and seemed to

be conscious. While being carried into a neighboring house, the convulsions reappeared, but compression of the carotids with both thumbs again caused the contractions to cease, and consciousness to return.

Epileptic convulsions had previously been controlled through digital compression by others. Binswanger speaks of it in his work on epilepsy as having been recommended by von Reimer in the middle of the last century and later by Corning. Hasse, according to Momburg (op. cit.), regarded the procedure as difficult, and had observed no successful trials of it. At the suggestion of Momburg, Volland was successful in several instances in controlling epileptic attacks by digital compression of the carotids. He observed, as did Hasse, that it is not always easy thus to compress the carotids, owing to the thick neck-musculature and the deep position of the vessels. My colleague at the Indianapolis City Hospital, Dr. Will C. Moore, succeeded in arresting the attack in a single instance by digital compression of the carotids.

Momburg permanently narrowed the common carotids in two cases with silver-wire loops, proceeding from the convenient hypothesis that in epileptics the seat of the disease is represented by the entire brain, and may be looked upon as an abnormal state of irritability, and that this irritability may be decreased by decreasing the blood supply and by decreasing the nutrition to lower the irritability of cells without affecting the vitality of such cells.

Momberg operated under novocaine-suprarenin anesthesia, compressing both carotids by twisting the crossed ends of a silver-wire loop until the temporal pulse became barely perceptible on the corresponding side. He operated on two cases, one in February and one in March, 1914. Distinct improvement followed in both instances. His published article in the *Deutsche*

medizinische Wochenschrift appeared within less than two months after the first operation and within one month after the second. It need hardly be said that a report made so soon after such an operation must be looked upon as of somewhat doubtful value for several reasons, two of which are that sufficient time did not elapse for the development of compensatory dilatation of the other vessels and establishment of collateral circulation, and, moreover, because such temporary beneficial results will, as has frequently been noted by neurologists, follow the performance of almost any operation upon an epileptic.

During August, 1914, I compressed both common carotids in two cases of general epilepsy, the plan followed being in most respects that adopted by Momburg. I did not employ local anesthesia in the first case for the reason that the attacks were so frequent, occurring as often as seven times a day, that I feared the onset of an attack might lead to a serious surgical accident. Silver wire, one millimeter in thickness, was passed under each common carotid, a short loop was cut off, and the ends were twisted until the temporal pulse on each side became so slight as to be just perceptible. A catgut strand was tied to the twisted ends of the wire, and left hanging out of the wound to be used as a guide in case threatening cerebral symptoms should require immediate removal of the wire. With the catgut strand, the wire loop can be drawn up into the wound, and snipped off with blunt-end scissors quickly and without an anesthetic of any kind.

Of these two first cases, one was in a young man of twenty years who had suffered five or six attacks of the grand mal type daily. The other case was that of a girl of seventeen, whose attacks, though much less frequent, were also of the severer form. Partial

compression of both common carotids produced in neither of these cases any untoward effects. Both patients slept soundly, as might have been expected in view of the decreased blood supply to the brain, for about eighteen hours. In both cases, the result, although gratifying, so far as the effect upon the frequency and character of the attacks is concerned, left much to be desired. The young man, though distinctly benefited, has had since the operation attacks of petit mal, in the average, one and one-half times a week. During the last month he has had but two attacks. Previous to operation the attacks, as stated above, occurred several times daily and were of severer form. In the case of the young girl, there has been a slight increase in the length of the interval between the attacks; and the character of the epilepsy has been changed for the time at least from grand mal to mild petit mal.

In the first week of November, 1914, I narrowed both common carotids with silver wire after the plan described above, in two additional cases. One was a case of petit mal in a man of thirty-five, the other a case of grand mal of the severest character and long-standing in a woman of forty-six. The man of thirty-five, with petit mal, was apparently somewhat improved, in that the interval between attacks was more than doubled in length. The severity of the attack was reduced. The result in the case of the woman of forty-five with grand mal was a complete failure. She developed coma immediately following the operation, and the silver-wire loops were removed, one within twenty-four hours after operation and the other four days later. Upon gradual recovery from the coma during the next two or three days following operation, a complete hemiplegia of the right side made itself

manifest with aphasia, from which the patient is now slowly recovering.

Three weeks ago I operated on a fifth case in the manner described. The patient was a man thirty-three years of age. He had suffered daily severe attacks following a blow upon the head over the upper Rolandic area on the right side. A decompression had been made without benefit. In the period of three weeks elapsing since the carotids were compressed he has had no attacks whatever. Since such a short period of respite may follow any imaginable operation upon an epileptic, such a temporary improvement may be said to be without great significance.

Momburg remarks that his results indicate with what safety the blood supply to the brain may be decreased. This is, I believe, slightly misleading, although it is frankly added that two observations cannot suffice for the drawing of general deductions. The one serious brain complication in my experience might be attributed, of course, to faulty technic or injudicious case-selection, yet it seems justifiable in view of the paucity of actual trials of carotid compression to say that, except in youthful persons, the sudden decrease in the blood supply to the brain may at any time, for aught we know, give rise to serious cerebral consequences. It should not be forgotten that the very factors upon which hope for success in carotid compression may be based,—namely, the meager facilities for the establishment of collateral circulation of the carotis communis and the restriction of the vertebrals in their bony canals,—represent factors of danger if the blood supply to the brain be decreased too greatly by the compression.

It is unfortunate that, in the case of the brain, it is not easy to determine by any of the known tests of the efficiency of circulation precisely how much change

is induced in the blood supply to the brain by compression of the carotids. Appreciating the great importance of such tests, the ignoring of which may lead to the incurring of serious and fatal errors in operations, one is, nevertheless, unable in the case of the common carotid to profit greatly by the employment of such tests. Matas (*Jour. A. M. A.*, Oct. 4, 1914), writing of circulation tests, remarks that "the time has now come when every progressive surgeon must regard it as his obligate duty to investigate and ascertain, by actual tests, what the behavior of the peripheral circulation will be whenever an operation is contemplated in which the parent vascular trunk is liable to be involved. This is no longer a matter of academic or theoretical interest, but a subject of practical, vital importance in the treatment of such cases."

The Pachon blood-pressure method, the test of the venous turgescence, the calorimetric test, and other methods of great value in determining the efficiency of the circulation in the extremities after occlusion of large trunks, are of doubtful practical usefulness in the case of the brain in comparison with the simple expedient of determining the strength of the pulse in the temporal artery. The excellent methods of Matas, particularly that of applying temporarily the aluminum band as a test of efficiency of circulation, would be of greater value in the cases under consideration were it not that the sleep, which is probably consequent upon the decreased blood supply to the brain, may mask cerebral symptoms which otherwise would give warning of danger.

In dogs, both common carotids and one vertebral have been ligated without fatal result; and in man, both common carotids have been ligated, one at a time, with only two weeks elapsing between the opera-

tions on the two sides. Stiles gradually occluded both carotids in hydrocephalus, two weeks elapsing between the beginning and the end of the occlusion. Lord (J. P.) and Frazier have also followed this gradual but complete occlusion plan in hydrocephalus with success, and Fenger (Ricketts, *Surgery of the Thorax)* completely ligated first one common carotid and then the other with an intervening period of a few hours, and with favorable results.

The favorable postoperative histories of the cases cited above indicate in what surprising degree the blood supply to the brain may be reduced in certain cases. However, it is by no means established that even gradual or partial occlusion of one common carotid is entirely free from danger of consequent cerebral mischief; and it need hardly be stated that partial occlusion of both carotids should be done with caution.

Instances of compression of the common carotids for epilepsy are so few in number that general conclusions must of course be withheld. In view of the extremely unsatisfactory results of non-surgical treatment of general epilepsy, the practice of the plan of compression of the common carotids may be considered justifiable, though the procedure at present should be regarded as an experimental one. Unfortunately, today, as centuries ago, we are reduced to the sad necessity of trying many uncertain remedies in epilepsy, trusting that at some time we may hit upon one that is truly efficacious. Certainly, carotid compression cannot be considered appropriate in cases in which there is the slightest reason to suspect irritation in any peripheral zone of inducing the attack, until such peripheral irritation has been excluded. If the operation is worthy of future trial, it is in cases of idiopathic general epilepsy, occurring in youthful persons, or in

traumatic or focal epilepsy which has persisted after cranial compression, or other appropriate direct intervention. In such cases the operation may be done without great fear of serious consequences and with considerable hope of benefit.

The circumstance that, in three of the five cases operated on by the writer, the severity of the attack was reduced, suggests that, whereas the increased influx of blood to the brain probably does not bear to the attack the relation of ultimate cause, nevertheless, the sudden influx of blood may determine, in some manner, the time and also the severity of the attack. Moreover, in view of the accredited fact that peripheral stimuli in almost any zone may induce epilepsy, it is still thinkable that the suddenly increased influx of blood to the brain may represent the agent which, activated by such stimuli, makes the direct assault upon the cortex. Obviously, an earnest effort to locate and correct any cortical lesion, or an existing peripheral source of irritation, for example, in a stagnant colon, should precede an attempt to reduce the aggregate caliber of the afferent vessels of the brain in general epilepsy.

DISCUSSION

Dr. John P. Lord (Omaha, Neb.): While I have had no direct experience in this manner of treatment of epilepsy, yet it appeals to me with particular force. It seems to me that this symptom, the tremendous cerebral congestion which accompanies these epileptic attacks, can be at least averted temporarily by this measure.

I have had some experience in the matter of ligation and compression of the carotids, and I wish to allude to the observation of W. S. Halstead, who speaks of the lessened mortality by the gradual compression of the carotids, notably in the treatment of aneurism.

The death-rate from immediate ligation of the carotid in aneurism, for instance, because of the peculiarly bad condition of the blood-vessels of these subjects, is at least 15 per cent

greater where it is immediately tied than if it is clamped off gradually by the aluminum clips.

I have ligated, in a number of instances, at two-week intervals, the carotids of children with hydrocephalus, according to the method of Stiles. Improvement was noted in all of them except one. In one I was satisfied that occlusion of the circulation took place so suddenly as to have produced a softening of the cerebrum. This child showed symptoms which indicated this, and it lived but a few months afterwards.

From the collected experience of others and from my own limited experience, I believe that this observation of Halstead has been abundantly corroborated by Matas and others. The gradual compression of the carotids, particularly the second one, is important; and it seems to me that in cases of epilepsy, which might be treated in this manner, it would be a much safer practice. The aluminum clip would reduce the circulation to what is thought desirable, and then at a suitable interval the operation of compression by the clip could be completed and thus obliterate entirely the circulation through the carotids, by which time collateral circulation would have taken place sufficiently, and thus perhaps reduce the percentage of risk in these cases.

My experience with hydrocephalus leads me to make this observation. I think it is rather an important feature, because I do believe there is a very material risk in ligating both carotids at short intervals.

My experience in the gradual compression of the carotids has been as stated. The procedure of Dr. Eastman answers the same purpose, of course, and it may be simpler to use, especially in the final stage.

Dr. Emil H. Beckman (Rochester, Minn.): I think it is pretty generally understood now that the gradual compression of the circulation is much safer than immediate ligation, because it allows the collateral circulation to become fairly well established before the main vessel is entirely obliterated.

I ligated at one sitting the external and internal jugular veins and the external carotid artery on one side on a patient, a woman, twenty-six years of age, with aneurisms in the head, probably venous and arterial. Ten days later I ligated the common carotid artery and the internal jugular vein on the opposite side. There were no untoward symptoms. Later, on this same patient I made an anastomosis between the spinal accessory and the facial nerve, for facial paralysis, which

apparently had resulted from erosion of the bone, due to this aneurism. This operation lasted over an hour, but the patient stood the operation without any trouble, and is in good health today, some two years after the ligation of these vessels.

Dr. Eastman (closing): I appreciate the discussion of Doctors Lord and Beckman, and particularly the remarks applying to the gradual compression of the carotids with Halstead's aluminum bands. I did not use these bands. It seemed to me that if it should be necessary to remove the constriction it would be simpler to untwist the ends of the wire than to push a knife-blade between the ends of the aluminum bands to separate them. Perhaps I ought not to express opinions about that, however, as I have had so little experience with the bands.

Removal of the wire was simple enough. We tied a catgut strand to the ends of the silver wire at the operation; and when we came to remove the loops we opened down to the wire following the catgut. It took only a minute or two to snip off the silver wire. I do not see how it could have been done any easier if we had used the aluminum bands.

Of course there is a wide difference between gradual compression and ligation. I do not think anyone would think of ligating even one carotid for epilepsy because of the mortality of that procedure, but I think in properly selected cases there should be a very low mortality in operations after the plan of Momberg.

It occurred to me to use the aluminum bands for this gradually increasing compression, but I decided not to use them for this reason: if we induce a very gradual decrease in the lumen of the common carotid we are allowing time enough for collateral circulation to be established, and we are allowing time for compensatory dilatation of the vertebrals. In other words, we are making no abrupt change in the circulation of the brain, so that the blood supply is maintained just about as it was before we began to compress at all.

That is, by the time you had sufficiently compressed your common carotids by the gradual method you would have ample collateral circulation; and you would not make any impression upon the brain by the change of blood-supply.

Although I have no proof to offer for the statement, it seems to me at least possible that, if by the partial occlusion as recommended by Momberg, we can secure a temporary cessation of the attacks,—that is to say, for a period of five or

six months,—perhaps after that the attacks would not return at all. We can never hope by such a procedure as Momberg's to change permanently the amount of blood which reaches the brain. There will soon be enough collaterals in young individuals and also compensatory dilatation of the peripherals. The hope is, that during the time when the amount of blood is affected by the compression the habit will have been cut off long enough so that it will not come back.

RECOVERY FROM PARALYSIS FOLLOWING DECOMPRESSION OF THE SPINAL CORD

Emil H. Beckman, M. D., F. A. C. S.

ROCHESTER, MINNESOTA

Surgery of the central nervous system is coming to be recognized as a branch of surgery in which the results may be compared favorably with those secured in many of the other special lines of surgery. Much of the haze of uncertainty which surrounded the diagnosis and treatment of diseases of the central nervous system has been cleared away by the brilliant light shed upon it by such investigators as Horsley, Sherrington, Cushing, Frazier, Spiller, Mills, and others. Investigation and experimentation have led surgeons of ability to attempt to cure by surgical means many lesions of the central nervous system formerly considered hopeless.

As a general rule, laparotomies and operations on the extremities which fail to reveal the pathologic lesion diagnosed or which disclose a neoplasm that cannot be removed, are likely to be considered as failures. Yet all surgeons are familiar with many instances of relief from symptoms and retardation of the growth of malignant abdominal conditions following an exploratory laparotomy. The rapid cure in tuberculous peritonitis that sometimes follows an exploratory laparotomy, is an illustration. In performing operations on the brain the same relief of symptoms, and an occasional cure when pathologic lesion is dem-

onstrated, are so common that the operation known as decompression is perhaps the commonest one performed on that organ. In this instance, however, the relief is believed to be due to the removal of pressure; yet in most instances no cerebral hernia develops, and the escape of cerebrospinal fluid must be of short duration. Is it not reasonable to suppose that the same factors that produce the improvement following an exploratory laparotomy may also assist here?

While the operation of decompression in brain surgery has become a recognized procedure, its application to the surgery of the spinal cord is more recent. The relief of symptoms following decompression of the spinal cord is at times as wonderful, and the beneficial results obtained are as surprising, as that obtained in cerebral decompression. When a diagnosis of tumor of the spinal cord has been made on a patient with root pains, level symptoms, disturbed sensation, and paralysis, and the operation fails to reveal the supposed tumor, it is truly humiliating to the surgeon; but if after such an experience the pain disappears, the paralysis leaves, and the sensation returns, the result is almost as gratifying to the surgeon as to the patient.

I wish to present two cases—one with no demonstrable lesion, the other with possible tumor—to illustrate the beneficial results that sometimes follow spinal decompression:

Case 1.—(A67505.) Male, married, aged forty-nine. Occupation, merchant. Examined May 6, 1912. Previous history, negative. Patient had suffered for several years with indefinite pain about the waist and down the thighs and legs. Occasionally the pain was severe for several days. He was operated on for appendicitis six years ago, during a periodic attack of pain which was believed to be appendicitis. He has had the same attacks many times since. One year ago weakness in the right leg was noticed, which disappeared in two or three weeks. Six months ago gradual weakness appeared in

the left leg. Five months ago there was some swelling in both legs. Three months ago the left leg became so weak a cane was used in walking; and for the past two months, because of the weakness in both legs, he has been unable to stand. He is now confined to a chair, and cannot move his legs about voluntarily. He suffers continuous pain in the lumbar region, which radiates around the body and down the thighs; there is involuntary contraction of the muscles of the thighs and legs, associated with pain when the legs stiffen. Some difficulty is noted in starting urine, but no actual loss of bladder control.

There is disturbed sensation below the level of the second lumbar vertebra, and heat and cold are not recognized below this level. The knee-jerks are much exaggerated. Ankle-clonus and Babinski's sign are present. The Wassermann reactions of blood and spinal fluid are both negative. Diagnosis: tumor of the spinal cord.

On May 16, 1912, laminectomy was performed, and the arches of the fifth, sixth, seventh, and eight dorsal vertebræ were removed. The dura was opened, but no lesion found. The diagnosis of tumor seemed so positive that on May 24 the arches of the tenth, eleventh, and twelfth dorsal vertebræ were removed, but no lesion was discovered except a small lipoma lying on the dura. This apparently caused some pressure, but could not account for the paralysis. The cord appeared normal, though congested. The wounds healed primarily. The patient suffered severely for several weeks from the spastic condition of the legs, which was associated with severe pain. At the present time, two years and seven months since operation, he is absolutely free from pain, has regained normal strength in the thighs and legs, and is able to perform all movements. He gets about on his hands and knees, and rides horseback. We have not been able to examine this patient, but from reports the only reason he is not able to walk is on account of the contractions which took place while he was suffering from the spastic condition. Undoubtedly these contractions could be relieved by orthopedic measures and he would be in a normal condition.

Case 11.—(A102880.) Single, male, aged thirty. Occupation, a wood worker. Examined March 23, 1914. Seventeen years previously, while playing football, the patient injured his back and was unconscious for about two hours, and had partial loss of power in both legs for two weeks, so that he was unable to walk. The bladder and bowels were not involved.

He entirely recovered, and was able to play football in a month. One year later he noticed a slight drag in his left leg, which seemed to be a loss of power in the muscles without any disturbed sensation. An orthopedist told him that the second lumbar vertebra was dislocated. At this time there was severe pain of a lancinating character, which extended down the spine from the lumbar region and into both sciatics. Three years later, in 1901, there was still some loss of power in the left foot, and he also had sudden severe pain in the back under the scapulæ, which seemed to produce a spasm of the muscles of the back. He had a temperature of 101° for three days, was in the hospital for six weeks, and part of the time morphin was required to control the pain. After that he was in his usual health until four years ago, when his left leg became weaker, and he had pain in the left hip and knee. From this time on he has continued about the same, with pain in both the left and right thighs, knees, and legs, with gradual loss of power of both legs. The bladder control is weak but not entirely lost. In December, 1913, the patient had sudden severe pain between the shoulders and the back of the head, which caused a retraction of the head. The diagnosis at that time was meningitis. The patient thinks he had some increase of temperature, but was in his usual health in two weeks.

Physical Examination.—Patient is small and thin. General examination was negative, with the exception of possibly a slight deformity at the first and second lumbar vertebræ. He is not able to walk, and is unable to lift either foot from the floor, although the feet and toes can be moved. The knee-jerks are exaggerated, and he has marked ankle-clonus. The spine seems rigid in the upper lumbar region. Tactile sensation, as well as the sense of heat and cold, is markedly impaired below the level of the second lumbar vertebra. The sensation is almost if not completely lost in the left thigh and leg, but not so much impaired on the right. The spinal fluid is negative, and the Wassermann reaction from the spinal fluid is negative.

On April 13, 1914, a laminectomy was performed, and the arches of the ninth, tenth, and eleventh dorsal vertebræ removed. The dura was tense, but not bulging. On opening the dura the entire canal was found filled with dilated blood-vessels which resembled an angioma. One of these vessels was accidentally punctured in opening the dura, and the hemorrhage was severe. In order to control this hemorrhage it was necessary to pack the opening in the dura with a strip of gauze.

The bleeding was easily controlled in this way. The dura was not stitched, but the muscles, fascia, and skin were closed with catgut. The gauze pack was removed on the fourth day, and the wound healed primarily. The patient remained under observation until May 20, thirty-seven days. Examination at this time showed that sensation was the same as before the operation. The bladder control has been almost perfect since the operation. There has been marked improvement in the motion of both legs; the patient can flex and extend the knees, and can stand alone, and walk several steps without support. The spasticity has also improved.

On June 20, sixty-seven days after operation, the patient wrote as follows: "I have not had any pain since I returned. I am improving nicely so far as gaining strength is concerned, as I am now able to go around without my crutches considerably and think I will be able to do away with them entirely in a short time. I have some twitching in my left leg, especially at night." On September 27, five and one-half months after the operation, the patient wrote: "I am doing nicely so far, and have been able to return to my work at last. I have not had a single pain of any kind except a burning sensation in my left thigh, which starts just above the knee and spreads over the front. There is also some slight twitching in my legs. This is only occasionally, and may be absent for several days at a time."

It is impossible to say whether these patients are cured or only temporarily improved, but the results have already justified the operation.

DISCUSSION

Dr. George A. Moleen (Denver, Colo.): In the presence of evidence of pressure within the spinal cord, decompression should theoretically offer a great deal. If it should be a tumor, however, the decompression is only temporary, as in a case which as yet has not been demonstrated, which was operated on in Rochester, and which had previously come under my observation, in which the symptoms were somewhat indefinite.

There were some symptoms of involvement of the posterior tracts; the muscular sense was not as well defined as it should have been; the reflexes were slightly altered as to degree; and the plantar reflexes were not certain, namely, at times suggesting extension and at others flexion.

The diagnosis was rather difficult. The report, as the newspapers had it, was of a miraculous recovery from a paralysis. The paralysis was not complete, however, when I observed the case, and was rather a disturbance in muscular sense than a loss of power. Upon inquiry it was stated that the case presented at operation a cystic tumor, and when the cord was incised a clear fluid escaped from a median-line incision.

The case at the present time does not show as complete a result as perhaps one might have looked for from spinal cord decompression. The case is now paralyzed, and all forms of sensation completely lost below the line of operation.

These are in direct relation, I think, with a good many attempts which have been made; and I do not think the results greatly differed from those of many operators, nor do I believe that they are so different from the attempts which have been made by Victor Horsley in removal of adhesions of the dura in cases of chronic pachymeningitis of specific origin.

Certainly, in cases of edema in the spinal cord, decompression should be of service, and also in those cases where we have definite neoplastic or hyperplastic dura. We should also remember that in cases of secondary or hyperplastic syphilitic meningitic or meningo-myelitic lesions we do have edema at the spinal roots, also the membranes are thickened in a great many of these cases, I believe, due to hyperplasia and edema of the membranes, especially around the posterior roots, and consequently there are the root pains from this source. It would be well, of course, if it were possible, to operate the entire length of the spinal cord under those circumstances; and in that way decompress the posterior roots, which are particularly, I think, the source of damage and the source of pain.

A great deal is to be gained from the modern refinements in diagnosis, and I think in all of these cases careful Wassermann reactions, confirmed by the lymphocyte count and the globulin reactions, should be done, and then we would know whether the hyperplasia was really due to a specific cause or whether it was due to some neoplastic cause. I think the edema has often been overlooked and a diagnosis of neoplasm made in the cases where the decompression has been done.

Dr. Beckman (closing): The more experience one has in the diagnosis of lesions of the spinal cord, as has been pointed out by Ellsberg, the more one finds that the classical symptoms

of tumor of the spinal cord are not always present, and that, in spite of the absence of certain of these classic symptoms, an operation is at times justifiable. Tumors of the spinal cord have been found in the absence of root pains and many of the other signs of tumor.

Of course, no one would advocate an operation of this kind without having made as accurate a diagnosis as is possible. But the point is, that, in spite of the most accurate diagnoses that can be obtained by present methods, there will be a great many mistakes, and consequently in some of these cases where there is a great deal of pain, where we are satisfied that there is a distinct lesion of the cord, with the symptoms localized to a definite area, an exploration is advisable, and, if the suspected tumor is not found, much relief may come from the decompression.

THE SURGICAL TREATMENT OF LARGE INGUINAL HERNIÆ

ALANSON M. POND, M. D., F. A. C. S.

DUBUQUE, IOWA

When Dr. Henry O. Marcy, of Boston, presented his paper on the principles of the formation of inguinal hernia, in 1870, he stated that he "felt sure that he had solved the thousand-year problem of the profession,—the permanent cure of inguinal hernia in the male."

Just how well Dr. Marcy has done his work, and just how thoroughly he has conducted his studies upon this great problem, can best be estimated when we observe that all operations devised by an exceedingly ingenious army of scientific workers are based upon the original work done by this eminent American surgeon.

There have been many variations of procedure to cure; many methods of the disposition of the sac; various methods of treating the cord; and numerous devices of closing the aponeurosis of the external oblique muscle: but there have been no material or important additions to the knowledge of the principles of the formation of inguinal hernia, or the factors to be considered in contemplating a cure.

The transposition of the testicle from the abdominal cavity, where it is formed, to the scrotal pocket, where it is to remain, takes an oblique course through the abdominal wall, from about a fingerbreadth above the middle of Poupart's ligament to the beginning of the

attachment of the scrotum to the lower abdomen. In adults this potential canal measures from two and one-half to four inches in length.

In the perfectly normal individual the intra-abdominal pressure is exerted at right angles throughout the entire extent of this oblique canal, hence the greater the pressure the more completely is the canal closed. In fact, the course taken by the cord is not a canal until a hernia exists.

The integrity of this canal, then, is determined by the ability of the tissues forming the posterior wall to press upon the tissues forming the anterior wall of this canal in varying degrees as exerted by the intra-abdominal tension.

The peritoneum does not figure in the consideration of any resistance, since the very presence of a hernial sac is due to the extreme elasticity of this membrane. The structure of the peritoneum investing a hernia does, however, undergo marked change in long-continued and neglected hernias. This will be considered later.

The posterior wall of the canal is formed by the transversalis fascia, which is just outside of the peritoneum and lies directly upon it.

Through this fascia the cord passes, and the internal ring is formed through it. This fascia forms the posterior wall throughout its entire length. At the inner third, or perhaps half, of the canal, the fibers of the internal oblique muscle, which at the outer portion of the canal lay anterior to the cord, wind around, from above, behind the cord, but in front of the transversalis muscle, and reinforce the fascia in forming the conjoined tendon just behind the external ring. This structure is dense and inelastic, but is quite incompetent to prevent a hernia unless the structures forming the anterior wall present a rigid support for it to

press against. The anterior wall is formed throughout by the aponeurosis of the external oblique muscle, through which the external ring is formed. In front of the cord, at its outer part, and anterior to the internal ring, the internal oblique muscle also strengthens this part of the canal for about a third of its course.

In the herniæ that have been unrestrained, the sac occupies the scrotum, because it is less resistant than the canal. But in the large herniæ that have been held with a truss or compression-pad, there have taken place many changes in the tissues that must be reckoned with when an operation for the cure of hernia is undertaken. The peritoneal sac, especially, has undergone marked change. Instead of a thin translucent membrane enveloping the intestines, it has become a tough, non-elastic sac, sometimes so dense as to resemble wet buckskin. The only trace of the original tissue remains in the lining of the tough sac. Here is found the delicate endothelial membrane exactly similar to the structure of the general peritoneal membrane.

The aponeurosis of the external oblique has also suffered change, which very seriously compromises the repair of the anterior wall of the canal. Long-continued pressure in the canal has attenuated the fibers of the aponeurosis, and the constant pressure of the truss pad or other means of support exerted on the outside of the canal, has seriously interfered with the circulation, until this aponeurosis has become a tissue of very low resistance possessing marked inability to repair, and the anterior wall has sagged until there is no support whatever for the posterior wall to press upon.

Hernias of this type are commonly found among the great class of industrial workers; men who have neglected the small hernias by reason of expense of time or money, and have tried to get along with truss

or pad until the increasing size of the hernia and the debility of years make the cure of the hernia imperative, or a state of invalidism imminent.

The operation selected for the cure of these cases should meet with certain requirements:

First. It should be as simple as possible, to permit of rapid operation and insure a permanent cure.

Second. It should seek to protect all the vessels and nerves supplying these tissues of acquired low vitality.

Third. It should offer a minimum of insult or damage to the two great factors underlying a cure of hernia, namely, the posterior and anterior walls of the inguinal canal.

Fourth. It should contemplate the obliteration of the sac and the repair of the relaxed anterior wall, in order to restore the integrity of the canal.

The operation to be described meets with these requirements, and has been used by the author for about five years with uniformly satisfactory results in thirty-seven selected cases, these patients ranging in age from 41 to 69 years of age. One case was operated on for a large double hernia.

To make the operative procedure perfectly clear, it will be convenient to divide it into steps as it progresses:

First Step. About two fingerbreadths inside of Poupart's ligament, an incision is made from a point where the internal ring is determined by deep pressure, paralleling Poupart's ligament throughout its entire extent and opening the upper third of the scrotum. This incision bares the aponeurosis of the external oblique muscle, and exposes the external ring and a part of the cord. (Fig. 1.)

Second Step. The sac is separated from the cord attachments in the scrotum, and freed from excess of fat with gauze by wiping the sac from the adherent

structures. The testicle is replaced within the scrotum, and the separation of the sac is continued up to and beyond the external ring. The excess of the sac may be removed if it is unusually tough or abundant.

Third Step. The sac is opened, and adherent contents are released and replaced within the internal ring.

A dry-gauze sponge, held in the bite of an eight-inch forceps, traumatizes the endothelial lining of the sac by vigorous rubbing over the entire surface up to and including the internal ring. This trauma destroys the protection afforded by the endothelial lining of the sac, and, in a very short time, permits of firm adhesion of the approximated surfaces. The sac is then twisted tightly, bringing the traumatized surfaces of the lining firmly together. (Fig. 2.)

Fourth Step. An eight-inch forceps is forced through the aponeurosis at the site of the internal ring, and passed downward within the canal, emerging at the external opening. The end of the tightly twisted sac is grasped in the bite of the forceps (Fig. 3), and is drawn up to, but not through, the opening made in the aponeurosis by the forceps. Here the end of the sac is anchored by a strong catgut suture. The twisted sac remaining as a strong cord within the canal. (Fig. 4.)

Fifth Step. Beginning at the upper end of the incision, catgut suture, armed with a round non-cutting needle, picks up a portion of the internal part of the aponeurosis, emerges to pick up the top of the twisted cord underneath the aponeurosis, and emerging again picks up an amount of aponeurosis on the external side to correspond with the amount taken up on the internal side. When tightened, this suture line brings two folded ridges of the relaxed aponeurosis together in the middle line and above the cord formed

by the twisted sac beneath the anterior wall of the canal. The amount of tissue taken up by this suture on either side of the twisted sac, must be determined by the laxity of the aponeurosis. (Fig. 5.) In cases where the hernia is very large and the anterior wall is consequently greatly relaxed, the amount of tissue taken by the lateral sutures will, obviously, be greater than in the cases of smaller hernia.

Care must be taken not to produce pressure upon the underlying cord and vessels, or edema will result.

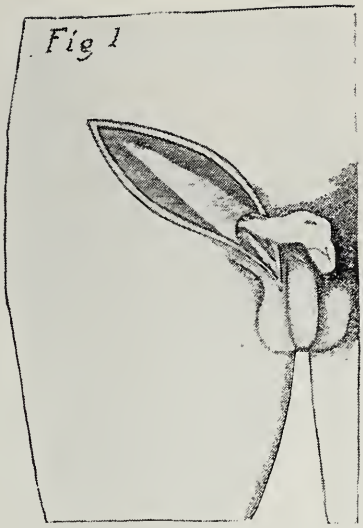
From four to six sutures are usually required to close up the relaxed anterior wall; and, when placed as described, the canal will be repaired by three distinct ridges, two being formed by the sutured aponeurosis, which rests immediately above one which is formed by the twisted sac.

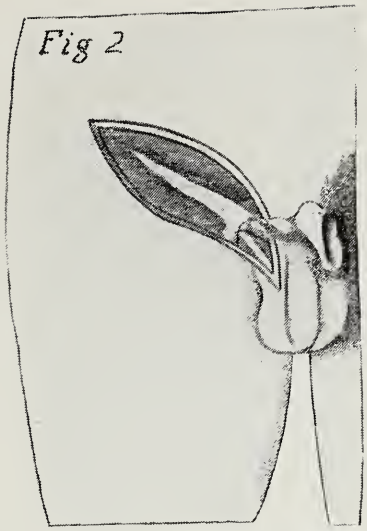
Sixth Step. The skin may be closed in any manner which is customary with the surgeon.

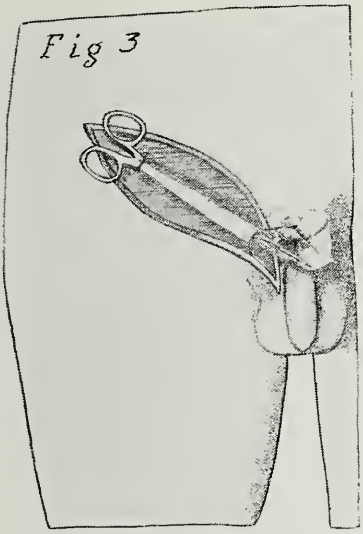
The operation permits of many minor variations. In two of my cases a small incision was made through the aponeurosis, and the cord and sac were drawn up in a loop through it, where the sac was isolated from the cord, and tied to better insure its obliteration.

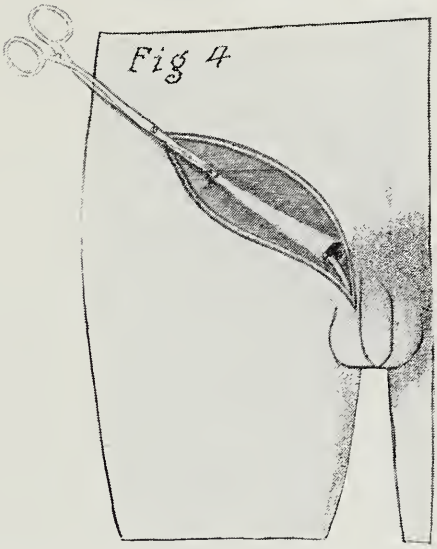
The traumatized peritoneal lining of the sac becomes adherent to apposing surfaces in a very short time, which not only obliterates the lumen of the sac, but, when twisted, forms a pad within the canal, which very materially lessens the capacity of the canal, and thereby increases the tension exerted by the intra-abdominal pressure of the posterior wall against the anterior wall. The suture line catches the relaxed aponeurosis on either side of the twisted cord, and anchors it firmly in the middle of the canal.

Throughout the entire operation there is no violence done to the nerves or vessels supplying the parts.









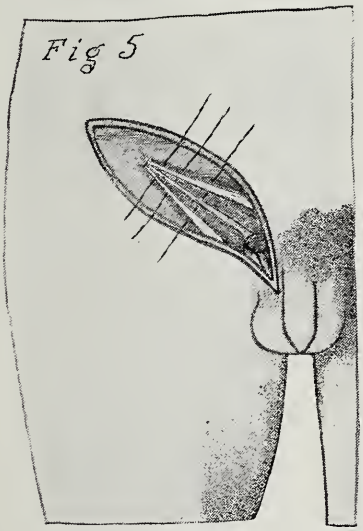


Fig. 1. The aponeurosis, external ring, and sac are exposed, the sac is withdrawn from the scrotum.

Fig. 2. The sac is opened; the contents are replaced in the abdominal cavity; the internal surface of the sac is briskly rubbed with dry gauze; and the sac snugly twisted.

Fig. 3.—An 8-inch forceps penetrates the aponeurosis and passes underneath it, emerging at external ring, where it grasps the end of the twisted sac.

Fig. 4. The sac is withdrawn underneath the aponeurosis, but does not emerge at the upper opening.

Fig. 5. The sutures take up the relaxed aponeurosis internally, passes through the twisted sac underneath the aponeurosis and takes up an aponeurotic ridge externally, completely closing the canal and taking up the relaxed anterior wall.

There is no incision through the attenuated aponeurosis of low vitality, which might later cause a recurrence by failure to heal. There is no need of a changed position of the cord.

This operation was devised to effect a cure of the large hernia of long standing in the industrial workers, especially. Prompted by a desire to take these men of advancing years from a state of semi-invalidism, and restore them to a point, near, if not quite, their full efficiency.

I have had but one complication that might be considered in any way serious. A case of an unusually large hernia, which had existed for a long time with a relaxed aponeurosis. In taking up the slack on either side of the replaced sac, too much tissue was taken, and edema of the canal and scrotum resulted. This edema gradually subsided, and the hernia has remained cured for more than a year at this time, the man being at work as a button-cutter, requiring him to stand from eight to twelve hours daily.

Traumatizing the lining of the sac, which permits of firm adhesions, the lateral placement of the twisted cord within the canal, which lessens the lumen and increases the possibility of pressure exerted by the posterior wall and the suture, which takes up the slack in the aponeurosis on either side and fixes the twisted cord in the middle of the canal, are the essential points of the operation; and, so far as I know, they have not been carried out in this way by others.

DISCUSSION

Dr. E. P. Quain (Bismarck, N. D.): If I understand correctly the technic employed by Dr. Pond in this operation for inguinal hernia, the reparative work is confined entirely to the anterior wall of the inguinal canal.

The chief reason for the presence of the hernia is to be found at the internal abdominal ring, and in the posterior wall of the inguinal canal. To tighten up the anterior wall

of the canal alone I cannot consider sufficient to insure permanency of result.

Nowadays local anesthesia permits us to operate on inguinal herniæ with comparative ease, even in grave and complicated cases. By means of it one can open the canal, and follow out whatever technic seems best for the repair of the posterior, as well as the anterior, wall of the inguinal canal. I should not feel that I had done justice in the treatment of such a hernia unless something had been done to close the deeper and more important defect in the abdominal wall.

Dr. Charles D. Lockwood (Pasadena, Cal.): It seems to me this operation is good. It is simply a modification of Kocher's operation, in which Kocher splits the canal and folds the tract upon itself, and so plugs the internal ring. The question of its value will depend upon the permanency of the results. If permanent in its results, it is an ideal procedure for large hernias in old people, because it does away with much trauma, and leaves the parts practically intact, with closure of the internal ring; but I have doubts as to the permanency of the results.

Dr. Pond (closing): First of all, I wish to emphasize a statement made in the reading of the paper, namely: This operation was devised to meet the requirements of repair in one class of hernia only, large inguinal hernia. It was not intended to become a routine practice to supplant or modify any of the recognized operations. It was intended to meet a specific indication in the cases of large inguinal hernia that could not be expected to hold where the aponeurosis was cut; and it was devised to avoid the cutting of this aponeurosis, which is the sole support of the anterior wall of the canal.

Replying to Dr. Quain, I will say that the folded and twisted sac within the canal acts as an anatomical pad, and thereby increases the tension exerted by the posterior wall against the anterior wall. The posterior wall has little, if anything, to do with the cause of an inguinal hernia and less to do with the repair. The cord and the sac come out of the internal ring, and lie above the posterior wall, which is formed by the transversalis fascia with some fibers of the internal oblique muscle.

I have used this operation for five years and in a series of thirty-seven cases. The first cases operated on in the series have not recurred.

In reply to Dr. Lockwood, I will say that Kocher's operation removes the sac from the canal and excises it outside of the aponeurosis, fixing the stump in the aponeurosis. He may invert the sac and withdraw it through the opening in the fascia; but in all his operations the sac is amputated, and the stump is fixed in the aponeurosis. The operation under discussion leaves the twisted sac within the canal; the trauma and the twisting completely obliterate the sac. It becomes then an anatomical pad, which fills up a part of the space occupied by the large hernia.

When the sutures are placed, the relaxed aponeurosis is taken up in two ridges which unite above the ridge caused by the twisted sac in the canal.

FACTORS DETERMINING MORTALITY IN PROSTATECTOMY

Charles D. Lockwood, M. D., F. A. C. S.

PASADENA, CALIFORNIA

The time has now come in the evolution of the surgery of the prostate gland when it is possible for surgeons to agree upon the fundamental points relating to the indications for operation, methods of diagnosis, technic, and postoperative care. To be sure, there is still a difference of opinion among surgeons doing a large amount of prostatic surgery as to whether the perineal or the suprapubic method of approach is preferable in prostatectomy; but I believe that most surgeons skilled in this operation would admit that essentially the same conditions prevail after a well-done perineal operation as after a suprapubic enucleation, and that the only important factor in the equation is the skill of the operator.

Statistics can now be cited showing from seventy-five to one hundred prostatectomies done consecutively by either the suprapubic or the perineal route without a death. The great improvement in the mortality statistics of prostatectomy in the last ten years, is due rather to greater care in preparation than to improvement in technic. However, there are several details of technic which in my hands have greatly increased the safety of the operation. Based upon an experience of about one hundred prostatectomies, the last seventy-five of which have been done without mortality, excluding carcinoma, I would designate the fol-

lowing factors as the most important in determining the safety of this operation:

No. 1. The preliminary examination to determine the operability of the case, and the probable resistance of the patient.

No. 2. The preparatory treatment.

No. 3. The anesthetic.

No. 4. The technic.

No. 5. The after-care.

No. 1. *The preliminary examination.*—This should be thorough, and should cover the following points: (1) A general physical examination, paying particular attention to the lungs, the vascular system, and the kidneys. Much can be done to lessen the dangers incident to high blood-pressure, bronchitis, or deficient kidney-function by the preparatory treatment. (2) Rectal palpation to determine the size, consistency and mobility of the prostate. The recognition of carcinomatous nodules will greatly modify the prognosis and will also determine the method of approach. (3) Determination of bladder-capacity and residual urine. Men who are otherwise bad risks, with good bladder-capacity and only a small residium, can often be rendered comfortable by judicious treatment without operation. (4) An *x*-ray of the entire genito-urinary tract. This will reveal vesical and renal calculi and pyonephrosis, and will obviate the necessity for using the cystoscope, which I do not believe is advisable as a routine in prostatic cases. It is difficult or impossible to introduce the cystoscope in many of these hypertrophied prostate patients; and pain, hemorrhage, and infection may follow its use. The information gained may be had by means of rectal palpation, catheterization and *x*-ray examination, supplemented by suprapubic cystotomy preliminary to removal of the obstructing gland. In a limited number

of cases operated on perineally, and in those having kidney complications, cystoscopic examination may be indicated. (5) Functional kidney test. I now make the phenolsulphonephthalein test in every prostatic case. It is conveniently done during the three or four days of preparation preliminary to operation, and while the permanent catheter or suprapubic drainage tube is in place. My experience with this test in prostatic hypertrophy has not led me to place great confidence in it as a guide to operability. While it is reassuring to have it appear within normal limits, as to time and quantity, I do not believe it should be a deciding factor in the determination to operate. I have several times operated successfully where there was an output of only twenty-five per cent in the first two hours. In determining the operability of the prostate, I have come to place more reliance upon the amount of urine eliminated in twenty-four hours and the specific gravity and the percentage of urea, than upon functional tests.

No. 2. *Preparatory treatment.*—This will vary somewhat with the conditions met with in the individual case. The margin of safety is often very narrow in these old men; and neglect of preparation may turn the scale against them. Almost every case is benefited by three or four days rest in bed, with a liquid diet and an abundance of fluids by mouth and per rectum. If the specific gravity is low with a large output, the bladder should be drained by a permanent catheter, suprapubic drainage, or systematic catheterization until the specific gravity rises to 1015, or more, and the urea output approaches normal. If the blood-pressure is above 160 it should be reduced, if possible, by rest, low diet, and catharsis. A dilated heart and bronchial râles should be regarded as grave complications. The presence of albumin in amounts greater

than can be accounted for by the pus and blood incident to a cystitis and coincident infection of the genito-urinary tract, are not serious complications, and will be largely overcome by the permanent catheter or suprapubic drainage. I do not believe there are any absolute contra-indications to prostatectomy, except a moribund patient and the absence of an expert operator. Nevertheless, the mortality will be in proportion to the care with which details are observed in preparation and technic. Another very important step of the preparation is to inspire these old men with confidence and hope. Many of them are discouraged, despondent, and ready to die. Vigorous assurance that they will recover, and enjoy better health than they have had for many years, will do more than heart-stimulants to fortify them.

No. 3. *The anesthetic.*—This is a most important factor in determining the outcome of these cases. For the past two years I have employed the anesthetic method designated by Crile, "Anoci-Association." The patient is given a preliminary hypodermic of morphia, gr. 1/6, and scopolamin, gr. 1/150. This eliminates all fear, and brings the patient to the operating-room in an apathetic and semiconscious state. Ether or nitrous oxide is then administered, preferably the latter, although owing to the expensiveness of nitrous oxide, we have not yet adopted it as a routine. The skin, fascia, and bladder-wall are now quickly infiltrated with novocaine, 1 to 400.

No. 4. *The operation.*—After opening the bladder, with prostates easily exposed, the prostatic capsule is injected with novocaine before beginning the enucleation. The adenomatous portion of the gland is now quickly shelled out with the index-finger, supported by two fingers in the rectum. This should be done quickly, but gently, never tearing or forcing the dis-

secting finger through the unyielding tissues. As soon as the tumor is delivered into the bladder, the prostatic nest, from which the gland has been removed, is gently compressed between the opposing fingers in the bladder and the rectum. The cavity quickly contracts until only the index-finger is admitted, and a firm, symmetric clot forms, closely simulating the enucleated prostate. In the last fifty cases, hemorrhage has never been alarming; and it is quite unnecessary to use packing or other device for controlling the bleeding. After removing the prostate with forceps, the bladder wall is thoroughly infiltrated with urea quinine, 1 to 600. A very large drainage-tube is now introduced, and the bladder wall tightly sutured about it with a pucker string of catgut, precisely as one sutures a gall-bladder. There should be absolutely no leakage about the tube. This will prevent infection of the prevesical tissues, with subsequent sloughing and delayed healing. No catheter is introduced into the urethra, and only gentle irrigation is employed through the large suprapubic tube. The fascia and skin are now infiltrated with urea quinine, 1 to 600, and the wound closed. The entire operation rarely requires more than thirty minutes.

No. 5. *The after-care.*—The tendency here is toward meddlesome surgery. Continuous irrigation and the urethral catheter and sounds I have abandoned, and my patients have made much better recoveries since. Too vigorous irrigation, and the pressure of a catheter in the urethra, tend to loosen the clot in the prostatic nest, and cause painful tenesmus. The passage of a sound or catheter is frequently followed by epididymitis. If the drainage tube is large and permits no leakage, and if the bladder wall, fascia, and skin have been well infiltrated with urea quinine, there will be practically no postoperative pain. In

comparatively clean bladders no irrigating is done until a few days before the wound closes, when the bladder is distended a few times through the suprapubic wound, to establish the patency of the urethra and to develop bladder capacity. In patients with a foul bladder, daily irrigations are necessary. The suprapubic tube is removed just as soon as clots cease to pass, usually the third day. The patient is gotten up by the end of the first week. The average time for healing is four weeks; many heal solidly in three weeks, and a few in two weeks. I have had two cases perfectly healed in ten days and passing all urine per urethram.

In the after-care, as in the preparation, I believe it is of the greatest importance to encourage the patient daily by strong assurances of his favorable progress and the certainty of a successful outcome. Sheer force of will, accompanied by confidence and unfailing good cheer, will snatch many of these old men from the slough of despondency, and place them upon the side of safety.

DISCUSSION

Dr. S. C. Beede (David City, Neb.): I am not sure that the essayist stated that the perineal method of operating is as desirable as the suprapubic; but among the factors which have lessened the mortality from enucleation of the prostate gland is the fact that the surgeons of this country have dropped quite largely the perineal route and adopted the suprapubic route.

The paper, I think, is very much to be commended. We often hear the advice given to close up the bladder in suprapubic operation, and let the drainage be through a catheter through the urethra. That is a mistake. A large drainage-tube should be placed in the bladder. Plenty of avenue of escape for the contents of the bladder is required.

The part in the paper which most impressed me was the non-interference after the suprapubic prostatectomy. If you want your suprapubic opening to remain open for a long

while, catheterize a few times. If you will absolutely give the urethra a rest after your suprapubic operation, the result will be very much better in the way of closure of the drainage wound, in the way of lessened contraction about the urethra, and in the way of lessened infection.

I think everything that has been brought out in this paper is very commendable, indeed, and I believe it expresses the best that there is in the present ideas of prostatectomy.

Dr. Lockwood (closing): In answer to the gentleman who just spoke about the choice of operations, I think perhaps he misunderstood me a little.

I believe that a few men who have the technical skill to do the perineal operation can do it with as low mortality as when it is done suprapubically.

Dr. Young has reported one hundred consecutive prostatectomies without death; and any one who can do Young's operation as he can do it can get just as low mortality as others can get by the suprapubic method.

Dr. Young told me the last time I was there what his statistics were, and I said, "You cannot beat that." He said he thought perhaps his operation was obsolete. I told him that it was not, in his hands, because he has developed a technic that makes it absolutely safe, just as safe as any man can do a suprapubic operation; and so I stated in my paper, that the question of the operator is the only factor that makes a difference in suprapubic and the perineal prostatectomy.

GASTRODIAPHRAGMATIC ATTACHMENT OF TUBAL PREGNANCY

Donald Macrae, Jr., M. D., F. A. C. S.

COUNCIL BLUFFS, IOWA

On September 24, 1914, the writer was called by Dr. V. Meyer, of Defiance, Iowa, to see the patient, Mrs. P. H., aged 29, and the mother of several children, the youngest six months of age.

Four months after the birth of her last child, the patient noticed a bloody discharge from the vagina, which continued to annoy her most of the time until September 23, when she experienced a severe pain in the lower abdomen, to be followed quickly by marked shock. The patient became blanched and pulseless.

On my arrival the following night, I found the woman in a state of semishock, but with a perceptible pulse and the history as given above. Upon vaginal examination I could detect a slightly boggy cul-de-sac. Thus far the way was clear, and a positive diagnosis of ruptured extra-uterine pregnancy was made. The woman had been pulseless; but, at the time I saw her, I could detect the pulse at the wrist, and decided against operation.

The foot of the bed was ordered elevated, and stimulant discontinued. Hypodermoclysis of normal salt solution was administered forty-eight hours later. Six days later the patient's physician reported marked improvement in the pulse and general condition, but that severe pain in the right chest and hypochondriac region had developed to an alarming degree. I, there-

fore, advised removal of the patient to the Edmundson Memorial Hospital at Council Bluffs.

Thus far I have occupied valuable time of this ultra-scientific body to record the classic symptoms of a simple case. Were I to stop here the sin would be unpardonable. My reason, however, for this report lies in the interesting features and operative findings to be developed later, coupled with the possibilities of the complicated and unusual terminal courses nature might have undertaken had surgery not interfered.

The patient arrived at hospital on September 30. Her condition was much improved; pulse, 154, temperature, 100° F.; appearance, white and waxy; respirations, rapid. She complained of great pain in the right liver and stomach region, with persistent vomiting. Physical examination revealed a moderately distended abdomen, with slight bulging in the upper right hypochondriac region, and here, upon deep palpation, the lower edge of an indefinite tumor mass could be detected. This mass seemed to be intimately associated with the liver, and extended over and apparently above the stomach. Much to my surprise, excepting the presence of an enlarged uterus, a pelvic examination was negative.

The writer had made a very positive diagnosis one week before; but at this time he saw visions of being forced to hedge He remembered Mr. Monyhan's experience in operating for ruptured tubal pregnancy only to find a ruptured duodenal ulcer. The mass in the upper right abdominal quadrant now looked suspisciously like a walled-off duodenal rupture; and yet I could not reconcile the coincidence of a large uterus with the intermittent flow. The blood-count, plus the appearance of the patient a week after the first shock, showed only too plainly that a severe hemorrhage must have occurred. Besides, the patient had vomited no

blood, her previous history pointing to gastric symptoms were negative, and, finally, at the hospital no occult blood was found in the feces. Therefore, I determined, in spite of anything to the contrary, to adhere to my first opinion, and decided to operate at the first favorable moment.

In spite of vomiting and pain, by means of rectal nourishment, salt water, and careful nursing, the patient regained much blood, and on October 19 the abdomen was opened.

The writer's left hand made straight for the pelvis, but much to his surprise found only a few adherent black clots. The left ovary and tube were normal, and the uterus enlarged. On arriving at the right tube, it was found to pass upwards and past the line of incision. The latter was extended upwards, and by aid of retractors the tube could be plainly seen passing from the pelvis upward along the inner aspect of the cecum and ascending colon. The fimbriated extremity was lost in a mass, which seemed to occupy a position posterior to and above the stomach, and attached to the under surface of the liver.

The condition, of course, was recognized as an unusual implantation or transplantation of an extra-uterus placenta. The mass was then quickly scooped out with the hand, the tube excised from the uterus, and the belly closed. In removing the placenta, fetus, and clot, the hand reached the esophageal opening in the diaphragm, a portion of the fundus of the stomach, as well as a considerable area of liver substance. The patient was discharged three weeks later in excellent condition.

Several interesting questions arise:

First. What possessed this tube to make such an excursion, and how did it accomplish the feat?

It does not seem possible that the mere raising of

the bed would answer the question and yet I have no other explanation.

Second. The stomach, being the only hollow viscus in contact with the mass and to all appearance about to rupture through the former, it is not improbable that, had surgery not intervened, this patient would have vomited a fetus.

Had this occurred in the writer's presence, the Western Surgical Obituary Committee would have had an extra-short report to make which, after all, might have been more acceptable than listening to this recital of "A Case."

RETROPOSITION OF THE COLON

Carl E. Black, M. D., F. A. C. S.

JACKSONVILLE, ILLINOIS

In 1911 I had the privilege of reading a paper before this Association on "Displacements of the Colon," especially discussing the great variety of positions assumed by the sigmoid. The experiences leading up to that paper greatly increased my interest in the practical importance of all matters relating to the colon, and especially in the wide variations in its position and the length of its several limbs.

I wish to invite your attention at this time to the history of a case which came under my care in March, 1912, as follows:

Personal history.—Female, single, aged 48, of excellent family and exemplary habits.

Previous illnesses.—History of obstinate constipation from childhood with many attacks of acute abdominal pain due to failure to get prompt relief from constipation. As she grew older these attacks became more frequent and severe, and were accompanied by indigestion with eructation of gas and a general neurasthenic condition. In 1910 she had an abdominal hysterectomy for hemorrhagic endometritis, which had existed for several years. She recovered without inflammatory reaction, and seemed much improved in general health, except that the attacks of constipation with abdominal pain and distention accompanied by nausea and vomiting, were more frequent and severe, and several attacks presented symptoms of acute obstruction, and then the contents of the intestinal canal would be slowly discharged, usually accompanied by considerable blood in the stools.

From the history it was evident that a narrowing of gut existed, and that the lumen was gradually growing smaller and

approaching a condition of complete obstruction, which did come on on March 4, 1912, making immediate operation imperative.

Operation.—Under ether anesthetic the abdomen was opened along the inner border of the right rectus. A large viscus was first encountered which proved to be an enormously dilated cecum, containing more than one gallon of fluid fecal matter. (Fig. 1.) There was complete obstruction of the transverse colon, a little to the left of its middle, from a twist in a direction which looked as though the cecum had turned forward from right to left through an arc of 180 degrees. The ileum, instead of entering the cecum on its left side, passed behind it, and entered on the right side, as it does before rotation. It was evident that originally the cecum had not descended into its usual position, as the ascending colon was comparatively short and the lower end of the greatly dilated cecum was not below the normal level. At the point of twist in the colon the duodenum passed in front of the colon, instead of behind it, and, while there was no narrowing of the lumen of the duodenum, it was thoroughly bound down to the colon by many dense adhesions, which were evidently of long standing.

The cecum was resected, and the ileum anastomosed into the transverse colon near its splenic flexure. The abdomen was closed without drainage, and the patient returned to bed in good condition. She did well for three days. The bowels moved, and she seemed almost out of danger when she died suddenly of cerebral embolism. No post-mortem was secured but she presented no symptoms which suggested ileus or peritonitis.

The important points observed were, that the position of the stomach was practically normal, there was no evidence of pulling or stretching of the stomach or duodenum, and the relation of the greater curvature of the stomach to the transverse colon was normal. The adhesions did not involve the stomach. The gall-bladder and ducts were normal, and free from adhesions, and no change in the relations of the stomach, duodenum, and bile-tracts was observed. The duodenal loop was fully developed in both length, diameter, and capacity, although it was perhaps not

quite as long as in the average subject; and there were no adhesions, except to the lower surface, where it crossed the transverse colon. The superior mesenteric artery ran a normal course. The cecum was quite free in the lower abdomen, being completely surrounded by peritoneum and having no adhesions to it to interfere with its mobility. On the right side there was a short transverse colon which was involved in numerous dense adhesions at a point where it passed behind the duodenum; it was difficult to distinguish an hepatic flexure, and to differentiate the cecum and ascending colon from the transverse colon, as it had no form of a true hepatic flexure on account of the enormous distention. On the left side the transverse colon was short, but otherwise normal; and the splenic flexure was well marked, although there were no supernumerary loops of colon in that region. The descending colon and sigmoid were normal. The flexure at the junction of the duodenum and jejunum was normal. All other organs of the abdomen were normal in position and condition. As far as could be observed there was a normal relation between the mesogastrium and the mesocolon. The only abnormality of the mesentery was its absence along the cecum and ascending colon, which were surrounded by peritoneum and freely movable in the abdomen. The colon was fully developed, except over a portion about six or seven inches long at the point of crossing of the duodenum. This portion was surrounded by dense adhesions, and had a constriction from a marked twisting of the gut producing complete obstruction. The small intestine, except the duodenum, was distended. The ileum passed beneath and behind the cecum, and entered it on the right side next to the wing of the pelvis. It was very easy to turn the cecum back into its normal position, allowing the ileum to enter from the left. The abnormality

seemed to be brought about by distention of the cecum. Apparently, as the cecum filled it rotated with the hands of the clock until the ileum assumed the fetal position and increased the twist of the transverse colon at the point where it passed behind the duodenum and caused the obstruction.

I have made a considerable search of the literature for similar cases, and there is certainly very little to be found. Among other things I wrote sixty prominent surgeons in the United States who operate in a large number of abdominal cases every year, and I have replies from most of them saying that such a case has not come under their observation. This of itself is sufficient proof of the rarity of the condition. I have found only two cases of retroposition of the colon reported about which there seems to be no question. These were reported by Tscherning[1] and Strehl[2], and the full reports are included in foot-notes.

NOTES

NOTE 1.—Case of Tscherning (Nord. med. Ark., 1883, xv, No. 9).—Quoted from Eckehorn.

A fifty-six year old workman (Fig. 16) died of ileus on the nineteenth day of his sickness. On autopsy the position of the intestines was found that is illustrated in Figs. 8 and 9. Directly midway between the umbilicus and the symphysis lay the cecum, which was very much distended. To the right above the jejunum and to the left the ileum, both markedly distended. It was impossible by pressure on the cecum to force its contents into the rest of the large intestine.

In Fig. 9 the small intestines have been removed. The cecum now goes back into its right place in the iliac fossa, and it is very easy to force its contents into the rest of the large intestine. We see that the duodenum has not finished the normal ring-shaped duodenal loop, but that it curves into loops like the rest of the small intestine, and is firmly adherent to the posterior abdominal wall. The colon in retroposition, the cecum and ascending colon freely movable to the right of the mesentery and spine, the rest of the large intestine in numerous loops to the left, completely empty and collapsed. The mesentery of the small intestine is fastened to the posterior abdominal wall along an S-shaped line, which begins above at the beginning of the jejunum and continues along the posterior abdominal wall to the end of the ileum. The ascending colon runs up to the boundary between the dorsal and lumbar vertebral columns. At this place it is crossed by the root of the mesentery. The root of the mesentery is adherent to the posterior abdominal wall, as well as below the colon, so that the colon bores through the base of the mesentery. No special transverse colon can be recognized. By the dislocation of the cecoascendans a transverse bend arose at the place where it passed through the mesentery.

NOTE 2.—Case of Hans Strehl (Archiv. f. Klin. Chir. v. 87, 1908, p. 8).

Child, Anna G. (See Fig. 17) twelve years old, from Fürstenwalde near Königsberg. Admitted August 17, 1907; died August 18, 1907.

Anamnesis.—Parents living and well; also several sisters. Malformation, as far as known, had not been met with before. The child was apparently quite normal.

Eight days before, she took sick with sudden severe pain in the lower right side of body without any outer cause; soon nausea and vomiting followed. The stools were arrested; from beginning to end but one stool was obtained.

Status.—A thin anemic girl with projecting abdomen. The breathing somewhat shallow and rapid. Pulse, 130; temperature, 38.5°; abdomen, all tumid and sensitive to pressure. Resonance throughout tympanic, but somewhat diminished in the cecal region. Liver dullness nearly abolished. Ill-smelling vomit, bilious masses.

Diagnosis.—Diffuse peritonitis, perforating appendicitis?

Operation.—A few hours after washing out the stomach with Billroth's solution (normal salt?) the body was opened along the right rectus muscle. Strongly filled and deeply congested coils of small intestine gushed out. After lengthening the cut above and below, it was found that the cecum and colon, distended to the width of the upper arm and filled with fluid contents, were discolored a blue-red. The appendix was normal. The lower end of the ileum was found in a similar distended state as the cecum. In following up the intestine, it appeared that the transverse colon without developing a hepatic flexure had passed under and behind an elbow, forming bridge of the mesentery of the ileum before the spine, and had been thus strangulated.

After turning over the whole small-intestine loops toward the right we found on the left the continuation of the transverse colon lying in a "coagulated" condition under the stomach in very many coils. Every attempt to change the position of the gut by traction failed, as the mesentery was fastened to the spine just beneath the opening, through which the colon passed. By the vain manipulation to replace, soon abundant, thick fluid feces were discharged upon the operating-table by the natural way. After careful cleansing, the intestine was replaced in the abdomen, the cecum was sewed widely into the abdominal wound to make a cecal anus on the next day. Tamponade. Suture of abdominal wound. The immediate opening of the artificial anus seemed unnecessary, as, after the spontaneous evacuation, the tension of the intestine seemed much relaxed. The child had also had several copious dejections. In spite of Koch salt infusion, etc., a rapid loss of strength ensued, and death occurred eight hours after operation.

For unrelated reasons only a hasty section was permissible; diagnosis at operation of a retroposition of the colon behind the ileum was correct; and it was a strangulation ileus through the mesentery of the ileum.

The abdominal viscera with the exception of the liver were excised in toto for further examination.

The stomach lies normally and presents no striking peculiarity. The duodenum had not the horse-shoe shape, but was grown into two upright loops, which lay one behind the other. It then passed directly without a distinct boundary before the colon into the jejunum. The pancreas lay, in normal size and position, behind the stomach and in the first loop of the duodenum, where, also in the customary place, the opening of the bile and pancreatic duct is found in the papilla of Vater. The small intestine in its shrunken state (alcoholic preparation) had a length of 3.5 m., the lower end of the ileum, about 30 cm., is much distended and livid. The mesentery of the small intestine forms a direct continuation of the mesentery of the duodenum, and continues as the mesentery of the cecum, so that one can speak of the common ileocecal mesentery. The colon at its beginning is very large, even as broad as the upper arm. The appendix shows no peculiarities; it is 8 cm. long and freely movable. The cecum is free only at its lower end; further up it is adherent to the spine. An hepatic flexure is wanting, as the colon is extended in a flattened arch from the iliac fossa to the spine.

Here it disappears under a sharp-edge bridge formed by the mesentery of the small intestine. Both pillars of the bridge are firmly attached to the spine. The muscle layer of the colon is greatly hypertrophied for about 10 cm. mouthwards from the strangulation. After the passage through thin hole, at the left of the spine, the transverse colon is "coagulated" and passed in many loops to the high-lying splenic flexure. This part of the transverse colon has a well-developed mesocolon, and is bound to the greater curvature of the stomach by the gastrocolic ligt.; also a short omentum is present. The descending colon, also empty, shows no peculiarities. Sigmoid flexure, normal; also rectum.

Measurements in alcoholic preparation:

Duodenum	23.	cm. long
Jejunioleum	350.	cm. long
Colon to strangulation	30.	cm. long
From strang. to sp. flex.	34.	cm. long
Descending colon	29.	cm. long
Sigmoid	30.	cm. long
Pancreas	15.	cm. long
At head	4.	cm. long
At tail	2.5	cm. long

The muscleature of the colon mouthwards from the kink is strongly hypertrophied for about 8 cm. There was in this case a false turning of the navel loop, which followed the hands of the clock, instead of turning in the opposite direction. We cannot tell what factors are to be regarded as the cause of the wrong turn in this case, because we have not been able to find a satisfactory reason for the turning in the normal direction.

I have found four cases which present some of the characteristics of this case, but about which there seems to be some question as to the actual condition or the cause or mode of origin. These were reported by Marchand[3], Boyd[4], Hausmann[5] and Clairmont[6]; and the full reports are given in the foot-notes. A number of other cases were examined but do not seem to properly fall in this group, and are not included, although references to them will be found in the bibliography appended.

NOTE 3.—Case of Marchand. Partieller "Situs Inversus" der Bauchorganen. In Ahlfeld. Berichte und Arbeiten der geburtshilflichgynekologischen Klinik zu Giessen, 1881-1882, p. 254, Leipzig, 1883.

Autopsy of a child born in asphyxia. On opening the abdomen it was seen that the stomach and spleen were not in the normal position. The course of the large intestine was far from normal, while the liver was normal with reference to position and relation of right and left lobes to one another. The stomach lay to the right beneath the liver, and appeared on lifting up the right lobe of the liver, the fundus was turned to the right, the pylorus to the left, the duodenum lying free in the middle. The small intestines were, for the most part, shoved to the left and upward, but could be displaced to the right; and when they were, a wide opening appeared behind the hepatico-duodenal ligament and the duodenum, through which the end of the little finger could be passed to the right. This opening apparently corresponded to the foramen of Winslow. The sac of the omentum could be blown up through it in the ordinary way, and the large omentum, hanging down from the stomach on the right, was also blown up.

The course of the large intestine varied extremely from the normal. On opening the abdomen it was covered for the most part by the small intestines lying in front of it; the cecum and appendix lay on the right, but very much displaced upward and toward the midline (about in the middle of the abdomen). There was no transverse colon to be seen, the sigmoid flexure lay to the left, but on a very long mesocolon.

The large intestine could only be seen after the small intestines were pushed upwards. The cecum, with the part of the large intestines corresponding to the ascending colon, hung free on a mesentery that was common to it and the small intestine; it was not fixed at any point to the abdominal wall. In the middle of the abdomen the colon made a sharp turn to the right, and then immediately back to the left, and in the course of this loop was bound fast to the posterior wall of the abdomen. Then it passed into the descending colon through a second small turn, which was not bound to the abdominal wall, but hung on a long mesentery which it had in common with the sigmoid flexure. These two last-mentioned loops were bound to each other by secondary adhesions, so that the parts of the large intestine could not be wholly separated from one another. Just in front of the spinal column this part of the large intestine was pushed upward so much that it lay immediately below the foramen of Winslow separated from it only by the root of the mesentery.

Note 4.—Case of Florence Nightingale Boyd. "Case of Intestinal Obstruction." (Lancet, July 3, 1897, ii, 8.)

Patient (see Figs. 14 and 15) was operated on for symptoms of intestinal obstruction, and died. She was a woman of 39.

Autopsy report: On opening the abdomen the most prominent objects were the much-distended cecum and the ascending colon, extending from the pelvis along the right side of the abdomen. Towards the left part of the tranverse colon was seen, and nothing else was visible but coils of small intestine, some being deeply congested. The cecum could be lifted out of the pelvis, and was quite free, being, as well as most of the ascending colon, surrounded entirely by peritoneum. Extending upwards on the posterior surface and bound down to it was the vermiform appendix. The liver was completely under cover of the ribs from the fourth to the seventh; the gall-bladder was adherent to the first part of the duodenum. The free cecum, ascending colon, and the greater part of the small intestines had made a complete revolution, from left to right and from behind forwards. It was this torsioned portion of the small bowel that was noted from the first as so congested. Continuous with the lower and left edge of the mesentery was a short, well-marked ascending mesocolon, the cecum and part of the ascending colon, as above stated, being quite free. The stomach was pale in color, of an hour-glass shape, and extending far down into the abdomen. On following it down, the pyloric end was found to pass downwards in front of, then upwards and behind, the right end of the transverse colon, the two viscera thus constricting one another.

The duodenum was next examined; the second and third parts bore the normal relation to the right kidney and pancreas, but the first part, owing to the faulty position of the pylorus, was too long and ascended vertically in front of the second part. The jejunum turned, as usual, sharply forwards and to the right; the peritoneum covering the first coil was adherent to the peritoneum covering the pancreas. The mesentery appeared at the upper border of the right end of the transverse colon in front of the junction of the stomach and duodenum and to the outer or right side of the colon. The origin of the superior mesenteric artery was normal. The small intestine was now removed. For twelve feet of its extent it was not injected; the remainder was of a purple color, and the mesentery of this part (the part involved with the cecum in the volvulus) was thickened, and contained swollen glands, which formed the firm mass felt before operation, the volvulus having brought it down behind and to the mesial side of the ascending colon, where it was found at the operation. There was a constriction just beyond the dilated hepatic flexure of the colon. The transverse colon here passed

in front of the vertically placed pyloric end of the stomach, and then from above down behind the stomach to gain its normal position with regard to the greater curvature. The splenic flexure was very acute, and there were adhesions between the transverse colon as it passed up to the splenic flexure and the descending colon. As the colon thus hooked round the stomach its caliber was diminished, and this seemed to be a cause of chronic obstruction. The author thinks that the reason the acute clinical symptoms appeared at this late date was that the volvulus had only recently occurred though the malposition was congenital.

NOTE 5.—Hausmann's Case. Beitrag zu den Lageanomalien, etc. Centralbl. f. Chir., 1900, p. 19. (Quoted from Eckehorn.)

This case was one of a twenty-two-year-old man who died of ileus. The common mesentery was about 14 cm. broad at the cecum, becoming narrower gradually toward the hepatic flexure. On the tranverse colon there was no free mesentery. The line of insertion of the common mesentery, the two folds of which lay in apposition to the base, ran in an arch, convex downward, the lowest point lying about midway between the cecum and flexure. The hepatic flexure lay at the normal level, and was contracted and narrowed for about 6 cm. The transverse colon was normal in appearance and form. On following it to the left it disappeared suddenly on the right side. The duodenum was firmly fixed to the transverse colon. No descending and ascending part could be distinguished in the duodenum, but it ran in a flat arch, convex to the right, downward and to the left, and after bridging over the transverse colon passed into the jejunum on the left side of the spinal column without any sharp bend. The transverse colon was fixed to the posterior abdominal wall throughout its entire extent without any free mesentery. The form and position of the stomach were normal. It was connected by the omentum with the part of the tranverse colon running left from the duodenum. The omentum was small in size and barely reached over the edge of the transverse colon, extending on the right to the duodenum. The part of the duodenum lying above the transverse colon did not lie directly against the posterior wall, but had a short, free mesentery.

If this position of the intestine, as described by Hausmann, took place from a volvulus, such as that described above, it must have occurred at a tolerably early stage. The transverse colon was at that time still free, but later, together with the hepatic flexure, grew fast to the posterior abdominal wall, while the ascending colon remained permanently free. The mesentery of the small intestine also grew partly to the posterior wall below the transverse colon. The 6 cm. long narrowing of the hepatic flexure may have been due to the twisting of the colon.

Hausmann gives the following explanation for his case:

The umbilical loop first underwent torsion in the normal direction, but the ascending limb, as it grew in length, turned not in front of, but behind the duodenum, the cecum then turned downward and continued its descent normally. Eckehorn urges against this explanation that, once the umbilical loop having undergone its normal torsion, there is hardly a possibility that the colon could get behind the duodenum in any other way than by a volvulus. Moreover in cases of true reposition there are hardly ever the normal three limbs of the large intestine.

NOTE 6.—Case of P. Clairmont. From a report of 258 stomach operations performed in the clinic of Professor v. Eiselberg. (Arch. f. klin. Chir. 1905, lxxvi, 212-316.)

In one of these cases, No. 153, the autopsy findings are given. The stomach was large and hypertrophic; on the anterior wall of the duodenum near the pylorus a white place formed of shining tendinous connective tissue. The transverse colon lay behind the duodenum, and there was compression at the point of crossing. On the anterior surface of the stomach at the greater curvature was a loop of small intestine lying over the transverse colon. The peritoneum of it was intact. The afferent limb of this loop ran tolerably horizontally to the right, to pass over into the small intestine lying beneath it. The efferent limb at

the site of the suture (a gastro-enterostomy had been performed) formed a downward hanging loop, and then passed into the duodenum. The transverse colon back of the loop was adherent with its hepatic flexure to the neck of the gall-bladder, then continued transversely to the left hypochrondrium, where there were adhesions to the spleen, and then formed a loop hanging downward from the left hypochondrium toward the right iliac fossa, the limbs of which loop lay close together, and were adherent to one another. The left ascending part of the loop formed by the transverse colon then passed over into the splenic flexure, where there were also adhesions. The large omentum, rather poor in fat, hung from the loop of colon, but seemed somewhat constricted above. On straightening out the colon it was seen that the upper horizontal part of the transverse colon ran behind the point where the duodenum passed into the jejunum, crossing over it. At the point of crossing, the duodenum seemed to be deeply constricted, so that its diameter was hardly 1 cm. The colon was also constricted at the point of crossing, and contained scybala.

This is a peculiar abnormality in the relative position of the colon and duodenum, which must undoubtedly be regarded as congenital. By the abnormal crossing a compression was exercised that probably favored the development of the ulcer. It explained why the gastro-enterostomy had not been successful, for, though the stenosis of the duodenum was overcome by it, the stenosis of the transverse colon persisted.

Cases where the cecum retains its earliest position in the left iliac fossa, as reported by Mayo and others, and cases of partial rotation of the cecum, as described by Huntington and others, may on opening the abdomen give an impression of "situs inversus."

Before taking up these cases I will call your attention for a moment to the question of the mode of origin. There are three authors who have written at some length on the subject, namely, Tscherning, Eckehorn, and Strehl.

According to Lewis (Keibel & Mall's Embryology) in a normal human embryo of 7.5 mm. the intestine has formed its primary loop (see Fig. 2-*a*), and the vermiform process is a part of its lower limb. By the time the embryo has attained the growth of 10 mm. this loop has rotated in a direction against the hands of the clock to such a degree that the lower limb has now become the superior limb (see Fig. 2-*b*). That part of the tube which is to constitute the large intestine is on the left side of the abdomen when this crossing takes place.

In private correspondence, Dr. Lewis suggests, "In order to produce the anomaly in question the rotation

must have occurred in the reverse direction (with the hands of the clock), so that the large intestine crossed the small intestine on the right side of the body (see Fig. 2-*c*). Thus it is not due to a failure or faulty rotation, but to a rotation in the reverse direction."

No embryo has been observed containing a loop twisted in the wrong direction, but as only three or four cases in adults have been recorded, so far as I am able to learn, this is not surprising. This explanation of the case I have reported seems the best and most reasonable one which has been offered.

In order to refresh the memory of the reader and to make plain the accepted view of the normal development of the intestine, I reproduce several cuts from Huntington's Anatomy of the Peritoneum. The first seven (Figs. 3, 4 and 5; Figs. 6 and 7; and Figs. 8 and 9) illustrate the several steps from the forming of the primary loop through the various steps in ascent, crossing rotation, and descent of the cecum and the other parts of the colon. The other three (Fig. 10 and Figs. 11 and 12) show the relation of the mesentery and the mesenteric arteries to these processes.

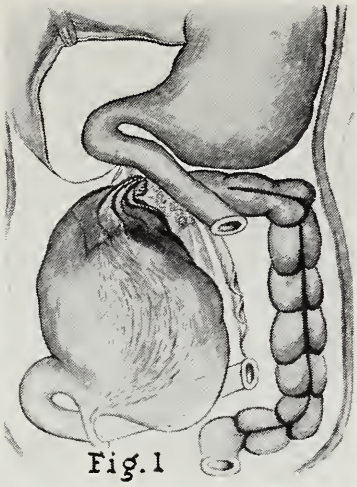
F. DeQuervain has written extensively on the subject of "situs inversus," and offers the following classification:

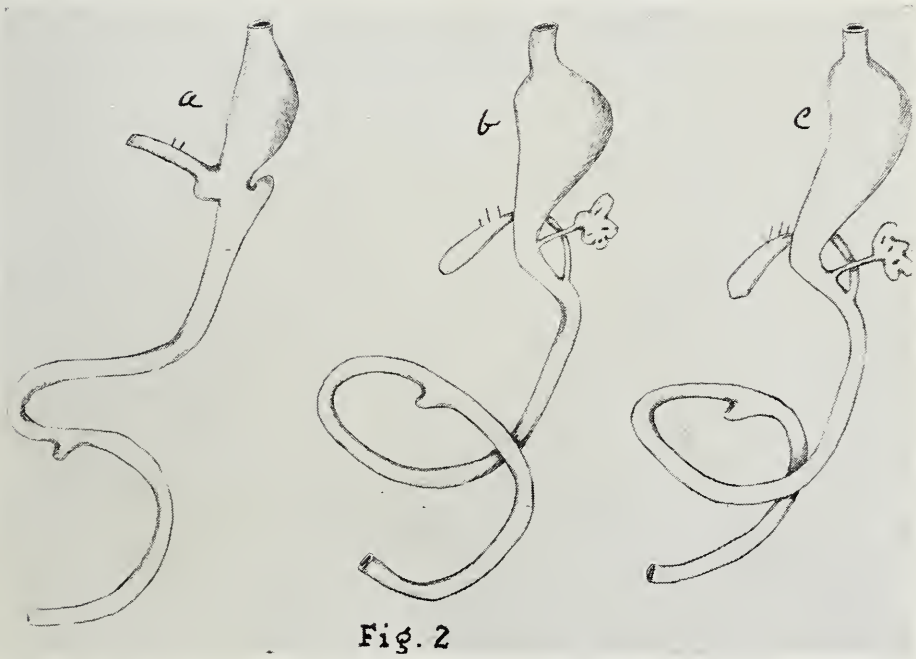
1. "Complete failure of the navel loop; backward location of the colon."

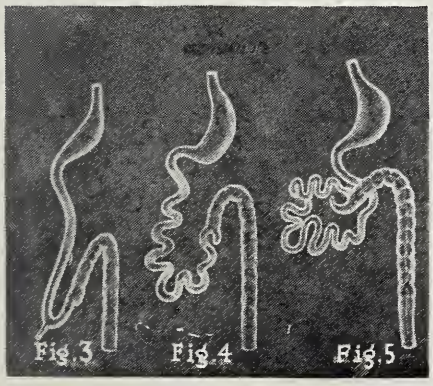
2. "Incomplete turning of the navel loop in the normal way; the colon lying towards the left."

3. "Incomplete turning of the navel loop in abnormal way (with the hands of the watch), the colon lying to the right."

4. "Complete turning of the navel loop in the direction of the hands of the watch; partial inferior 'situs inversus.'"







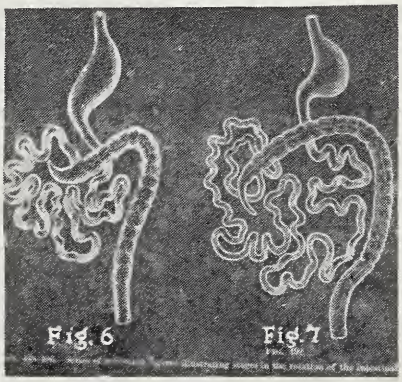
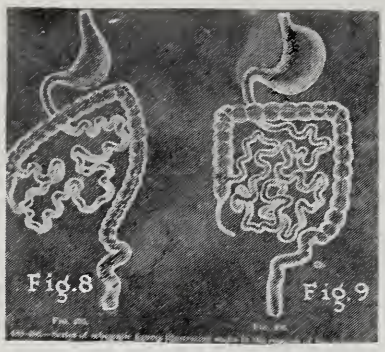


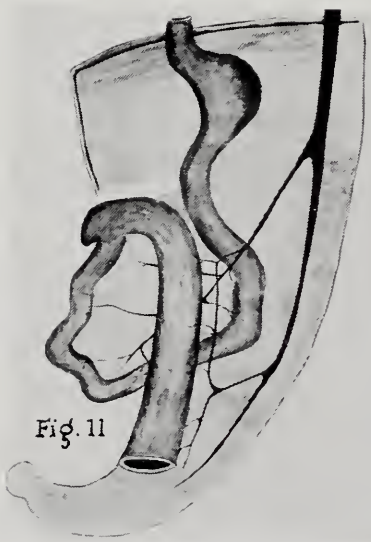
Fig. 1. Author's case. Duodenum passes in front of transverse colon. Cecum and ascending colon rotated to fetal position. Complete obstruction from twist of transverse colon.

Fig. 2. Torsion of primary loop. *a*, primary loop; *b*, torsion of primary loop, against hands of clock (normal); *c*., torsion of primary loop, with hands of clock (abnormal—may explain author's case).

Figs. 3, 4 and 5. Schematic figures illustrating early stages of rotation of the intestinal canal.—After Huntington.

Figs. 6 and 7. Schematic figures illustrating intermediate stages of rotation of intestinal canal.—After Huntington.

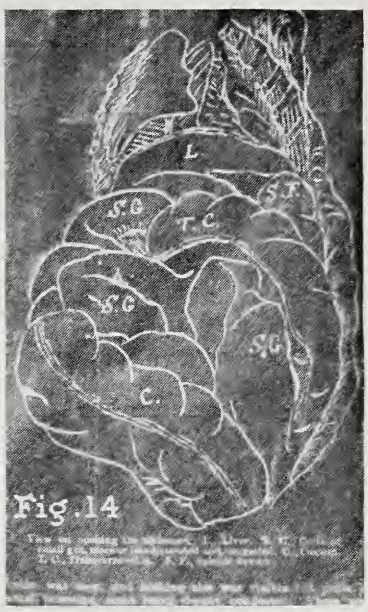


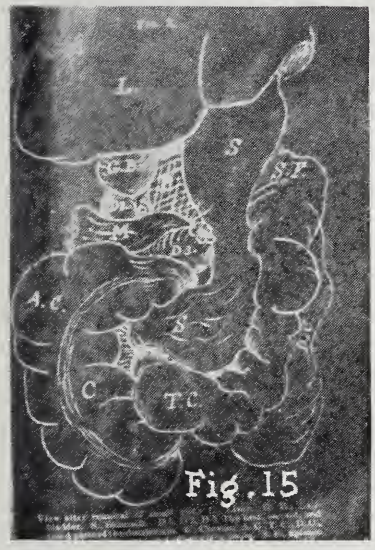


Figs. 8 and 9. Schematic figures illustrating later stages of rotation of caecum.—After Huntington.

Fig. 10. Schematic representation of umbilical loop, mesenteric attachments, aorta, and superior and inferior mesenteric arteries in an embryo of six weeks.—After Huntington.

Figs. 11 and 12. Schematic representations of the umbilical loop, mesenteric attachments, aorta, and superior and inferior mesenteric arteries during rotation.—After Huntington.





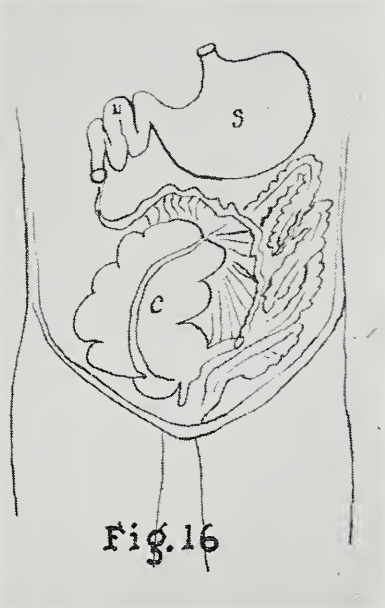
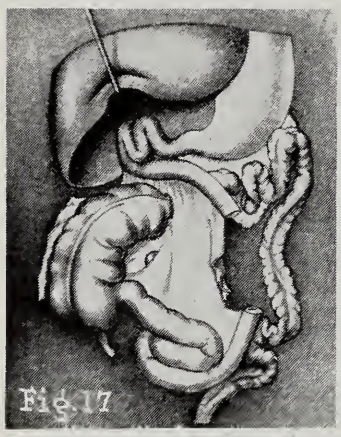


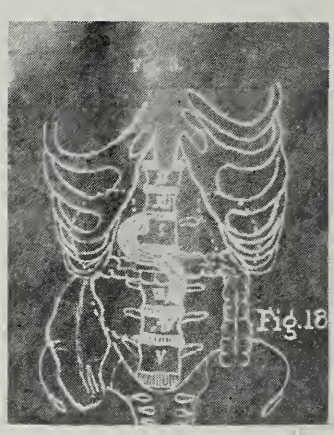
Fig. 13. Schematic retroposition of the colon.—After DeQuervain.

Fig. 14. Boyd case of retroposition of colon from volvulus. *l.*, liver; *s. g.*, small gut; *c.*, cecum; *t. c.*, transverse colon; *s. f.*, splenic flexure.

Fig. 15. Boyd case of retroposition of the colon from volvulus, after removal of small gut. *L.*, liver; *g b.*, gall-bladder; *s.*, stomach; *a. c.*, ascending colon; *t. c.*, transverse colon; *d. c.*, descending colon; *s. f.*, sigmoid flexure.

Fig. 16. Tscherning case of congenital retroposition of colon. Small intestines removed.—After Eckehorn.





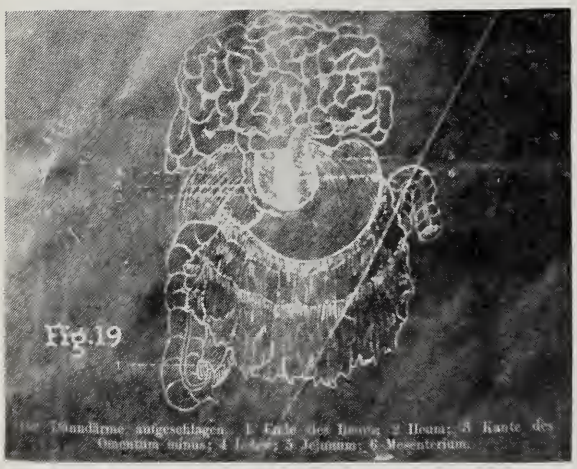


Fig. 17. Strehl case of congenital retroposition of colon. Small intestines removed.—After Strehl.

Fig. 18. Hausmann case classified by Eckehorn and Strehl as a volvulus; may have been a congenital retroposition of the colon.—After Eckehorn.

Fig. 19. Krafts case of volvulus simulating congenital retroposition of the colon. 1, end of ileum; 2, ileum; 3, small omentum; 4, liver; 5, jejunum; 6, mesentery.—After Eckehorn.

Fig. 20. Sundberg case of volvulus simulating congenital retroposition of the colon.—After Eckehorn.

My case would seem to belong to DeQuervain's fourth classification.

Eckehorn thinks that in these cases there are certain abnormalities about the duodenum which predispose to its anterior position. He suggests the following conditions in support of this theory: The duodenum (a) does not take so complete a ring shape as normal; (b) does not cross the spine at its ascending portion; (c) has no inferior horizontal part to pass over into the jejunum; (d) no jejunal flexure; (e) the duodenal loop is fully developed much earlier than the ileocecal loop, and this powerfully developed loop may be the cause of torsion of the umbilical loop; and (f) the normal development of the duodenal loop makes complete torsion of the umbilical loop a necessity.

These views are largely speculative, and the simpler view advanced by DeQuervain, Strehl, Lewis, and others, is much more to the point. (See Fig. 2-*a, b, c.*) There have been so few cases reported that the cause is necessarily involved in doubt. If we accept De Quervain's view (see Fig. 13) that a true retroposition of the colon consists in the entire large intestine being placed behind the small intestine none of these cases would answer to the definition.

The theory of a volvulus as the cause of such a case as I have reported, is impossible. Some cases which have been reported as retroposition of the colon are undoubtedly cases of volvulus coming on after development is complete. In my case, and the other two cases found in the literature of unquestioned retroposition of the colon, there seems no reason to think they were caused by torsion of the stomach around the umbilical loop as suggested by Boyd (see Figs. 14 and 15) and Faltin.

Eckehorn furnishes the following explanation of the Boyd case:

"After a normal torsion of the umbilical loop the stomach and intestine developed in the usual way and in the normal position. But the fixation of the entire ascending colon took place, and probably more of the right part of the transverse colon. A loop of small intestine now got behind the free ascending and transverse colon from below, rose through the dorsal mesogastrium behind the stomach, then through the lesser omentum to the surface of the stomach. Later, this pulled all the rest of the small intestine and the free part of the colon after it, and this produced both the intertwining of the stomach and intestine and the volvulus of the packet of small intestines and the free part of the colon, by which the transverse colon came to lie distally and dorsally from the root of the mesentery and the duodenum. In order for this to happen there must have been a defect both in the lesser omentum and in the dorsal mesogastrium. Such defects have been observed in several cases."

This explanation only shows that the case was not one of true retroposition due to reverse rotation during development, but one in which a number of the defects of development are involved. I present cuts of the Tscherning case (Fig. 16) and the Strehl case (Fig. 17). I regret very much not to have been able to secure a better cut of Tscherning's case, but I trust the description will supply the deficiency.

According to Eckehorn the fetal position of the cecum is a part of the process, and not as appears in my case an incident of distention, permitted by the absence of fixation of the cecum and ascending colon. This does not seem to have been the condition in either of the other cases. The abnormality was plainly not due to faulty development of the colon. The colon in each case was fully developed, except at the point of constriction, with the possible exception of a short

ascending colon and a somewhat imperfect hepatic flexure; but this form of hepatic flexure is not uncommon, and can hardly be considered an anomaly of development, but rather a defect in growth. And, again, if we would accept Eckehorn's view, the case reported could not be considered one of congenital retroposition of the colon because the transverse colon and greater curvature of the stomach maintained a normal relation. While Boyd's case was one of volvulus it was so different from the three more typical cases reported that it does not seem to me to affect the discussion. The same thing is true of the cases reported by Marchand, Toldt, Tandler, Kraft, Sundberg, and Clairmont. The classification of the Hausmann case (see Fig. 18) is still in doubt but probably should be classified as a true retroposition. While Eckehorn and Strehl are inclined to consider Hausmann's case as one of volvulus, Eckehorn modifies this view by saying, "It must have occurred at a tolerably early stage."

Hausmann gives the following explanation of his case:

"The umbilical loop first underwent torsion in the normal direction, but the ascending limb, as it grew in length, turned, not in front of, but behind, the duodenum; the cecum then turned downward and continued its descent normally." Eckehorn urges against this explanation that, once the umbilical loop having undergone its normal torsion, there is hardly a possibility that the colon could get behind the duodenum in any other way than by a volvulus. He also urges that in cases of true retroposition there are hardly ever the normal three limbs of the large intestine, which is apparently disproved by each of the three cases reported. Some of the other cases referred to are so plainly not of this class that it is hardly worth

while to introduce them in the discussion. At least two of these, Kraft's case, reported in the *Nord. Med. Ark.*, 1900, No. 13, and the Sundberg case reported by Eckehorn in the *Ark. f. klin. Chir.*, 1903-1904, are worthy of further study and cuts (Figs. 19 and 20) are included for that reason.

In the main the two cases cited in addition to my own case present practically the same picture and must have been developed by the same cause and seem to belong to a class peculiarly their own, which, while not answering the classical scheme of DeQuervain (see Fig. 13), we are probably justified in classifying as cases of "retroposition of the colon." The practical interest in such cases arises from the difficulties which abnormalities of the colon offer to correct diagnosis and proper surgical procedure.

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SOME OBSERVATIONS ON MESENTERIC OCCLUSION

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The bibliography on this subject is not extensive. Virchow first described the pathology in 1847. Welch carefully reviewed the subject up to the year 1900. As many as seventy cases of mesenteric thrombosis and embolism had been reported at that time. Gerhart and Kussmaul in 1864 had studied the clinical features. Watson and Elliot, in 1895, considered fifty cases in analysis from a surgical standpoint. Welch and Mall, Litten, Faber, and others have studied mesenteric occlusion experimentally. The work of Welch and Mall seems the most thorough and satisfying.

Since the year 1900 there have been several reports. Jackson, Porter, and Quimby reviewed 214 cases in 1904. Elliot's resection of four feet of infarcted intestine and Gordon's resection of two feet of infarct are the only two cases successfully operated on up to the year 1900. Jackson's, Porter's, and Quimby's reports note four cases successfully operated on out of forty-seven operations. Wakefield, in 1911, reports a successful resection of thirty inches of intestinal infarct.

The establishment of collateral circulation in most parts of the body seems easily accomplished. This is a wise and beneficent provision of nature. The mesenteric and intestinal anastomosis is larger and freer than that of any other part of the body. It seems that

here we should have most easily established a compensatory collateral circulation. If we eliminate all factors, except merely mechanical occlusion, we no doubt would have a collateral circulation as easily established as elsewhere. The modifying factors are, first and most important, the presence of infection. The other factors are the amount of mesentery occluded and the suddenness of the occlusion.

Thrombosis of either veins or arteries produces hemorrhagic infarct and consequent necrosis. Almost uniformly it is an hemorrhagic infarct. Faber has attributed the infarct to back-pressure in the portal system being greater and overcoming the pressure from the anastomosing vessels. However, hemorrhagic infarct results as readily when the mesenteric vein is thrombosed or tied. The studies of Welch and Mall are most rational and seem to furnish the best explanation of the phenomena. Their conclusions from experimenting on dogs were, that the blood producing hemorrhagic infarct enters by anastomosing vessels, and not by reflux from the veins, and that infarct is not due to demonstrable changes in vessel walls, but to retardation and stasis of the circulation, clumping the red cells in veins and capillaries and diapedesis. Another factor also is loss of lateral pulsation in the vessels. Perhaps a still more important factor is the violent tonic contractions of the intestinal walls, which takes place when a vessel is occluded, producing ischemia for two or three hours, followed by relaxation and infarct. Now, why conclude that infection is the most important element entering into the establishment of hemorrhagic infarct of the intestine and mesentery? If to ischemia, followed by dilatation and engorgement of vessels, stasis of current, clumping of cells, and diapedesis, we add infection with consequent changes in the vessel walls, we ac-

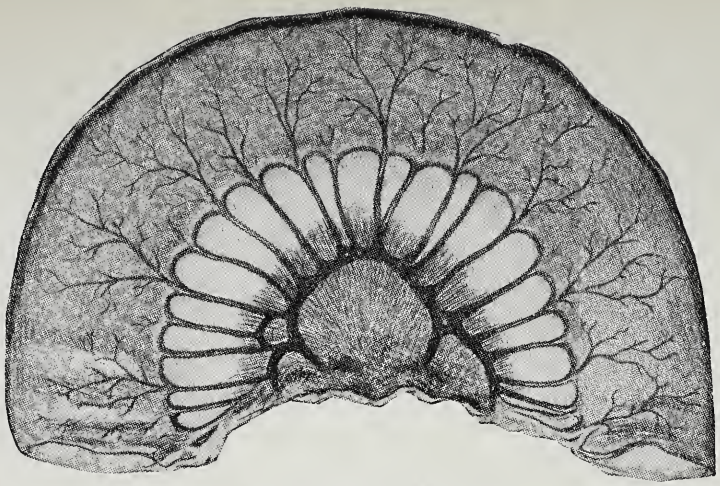


Fig. 1. Three feet from end of duodenum. Intestine twenty-three feet long (Monks). Note the system of loops formed by the branches of the mesenteric artery—primary, secondary and even tertiary. The vasa recti also provide very free communication between the terminal vessels on the surface of the bowel.

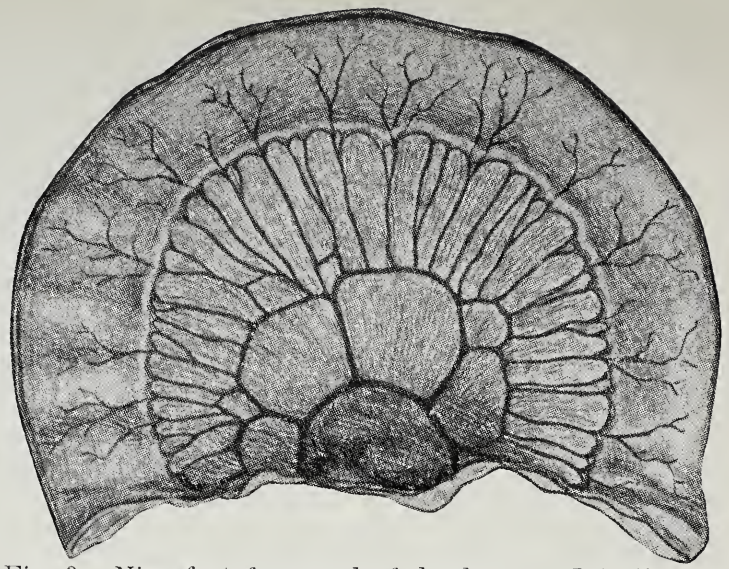


Fig. 3. Nine feet from end of duodenum. Intestine same as No. 1 (Monks).

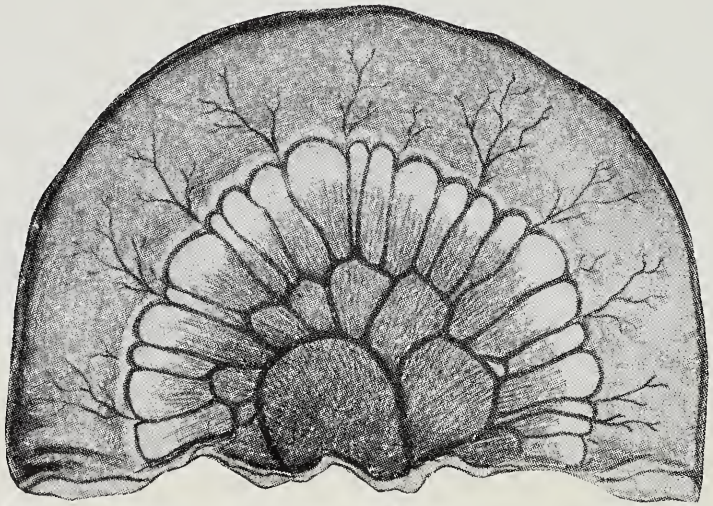


Fig. 2. Six feet from end of duodenum. Intestine twenty feet long (Monks).

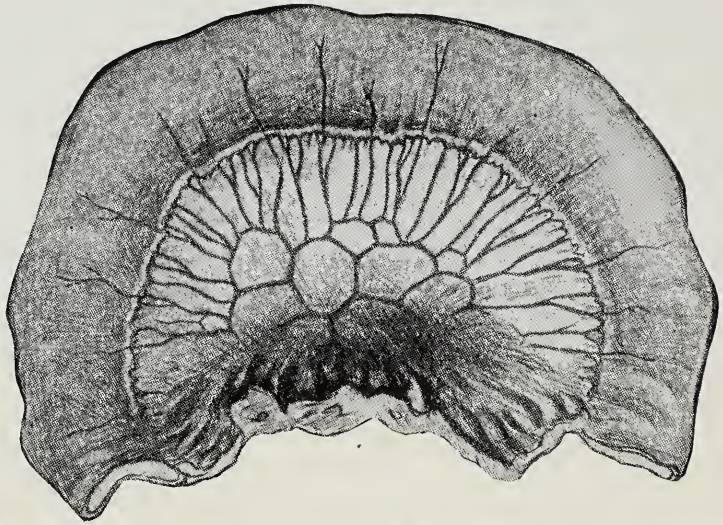


Fig. 4. Twelve feet from end of duodenum. Intestine same as No. 1 (Monks).

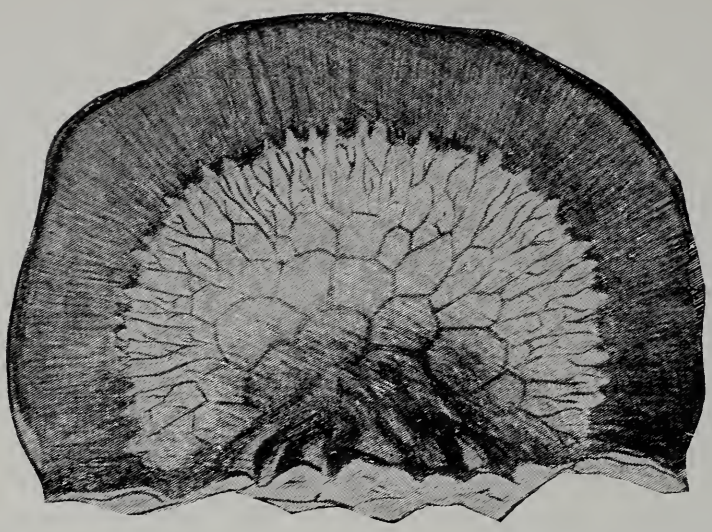


Fig. 5. Seventeen feet from end of duodenum. Intestine same as No. 1 (Monks).

Fig. 6. Twenty feet from end of duodenum. Intestine twenty-one feet long (Monks).

Photograph No. 1 taken before ligation.

Photograph No. 2 taken 15 minutes after ligating.

Photograph No. 3 taken thirty minutes after ligating.

Photograph No. 4 taken ninety minutes after ligating.

Photograph No. 5 taken twenty-four hours later. No pulsation in the tied mesentery, but no infarct. Abdomen closed again. Seventy-two hours later dog died. No marked infarct or gangrenous intestine. Peritonitis and adhesions.

Fig. 7. Dog. No. I. Ligature around mesenteric branch near base. Pulsation ceased in two-thirds of the mesentery from cecum upward.

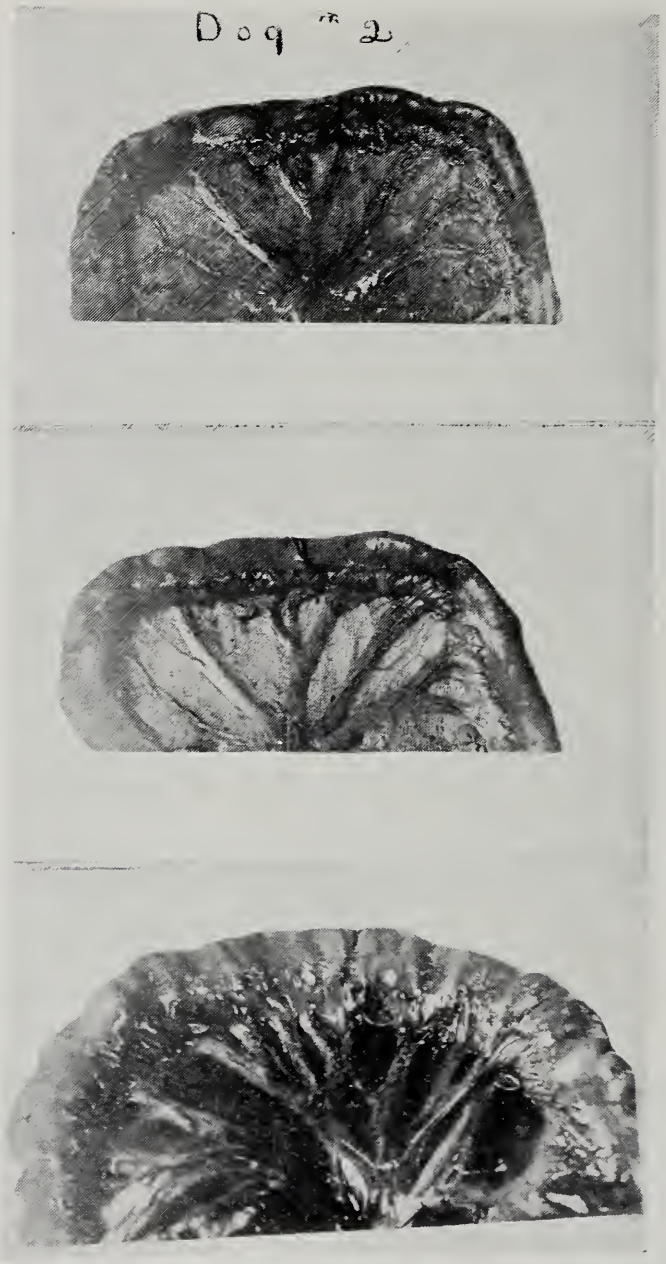
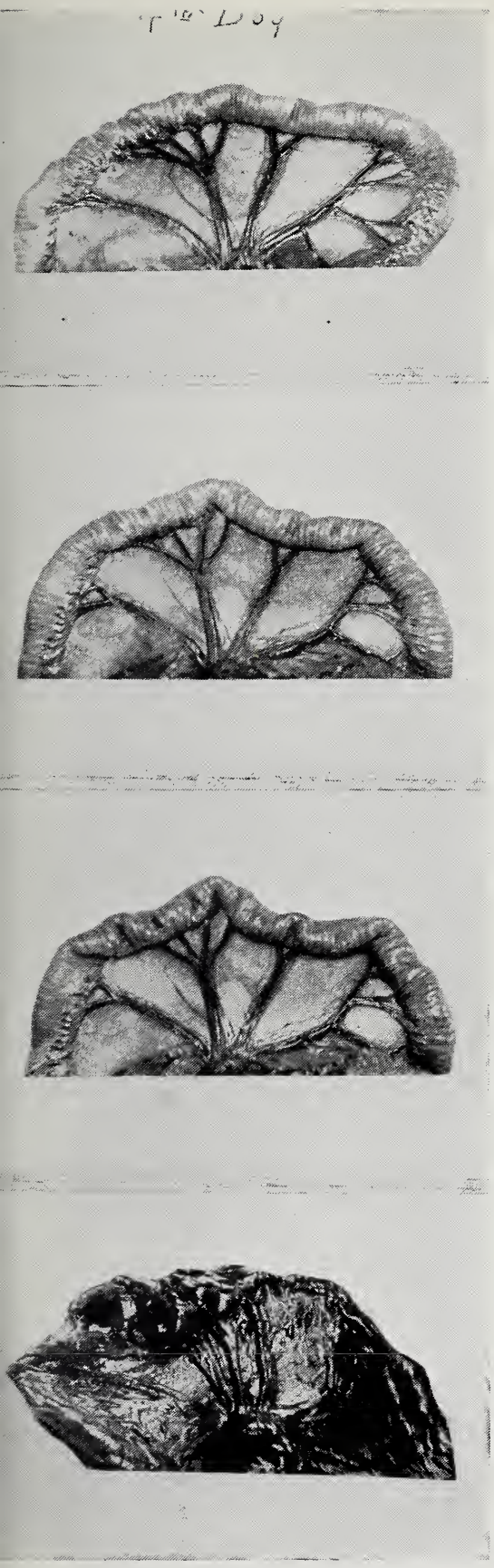


Fig. 8. Dog No. 2. Tied five vasa recti about one foot from the ileocecal valve.

Photograph No. 1, taken fifteen minutes after opening abdomen and five minutes after tying vessels.

Photograph No. 2, taken thirty minutes later and abdomen closed. This picture shows a marker indicating where visible pulsation ceased about two inches to the right of the vessel which was not tied.

Photograph No. 3, taken after opening the dog nine days later. No infarct and no gangrene. Intestine warm but without visible pulsation. Collateral circulation seems to have been established. Ether forced to kill dog. Noted after death for thirty minutes the slow peristaltic contraction-wave produced by mechanical irritation. From the point of irritation the wave slowly extending downward.



Photograph No. 1 before tying.

Photograph No. 2 taken fifteen minutes after tying.

Photograph No. 3 taken thirty minutes after tying.

Photograph No. 4 taken of infarct. The dog died about thirty-six or forty hours after time of opening. Infarcted mesentery in the lower two-thirds. The infarct was more extensive than the pulsation occluded from the ligature at first. Dog had tarry stools and bloody serum in peritoneal cavity.

Fig. 9. Dog No. IV.

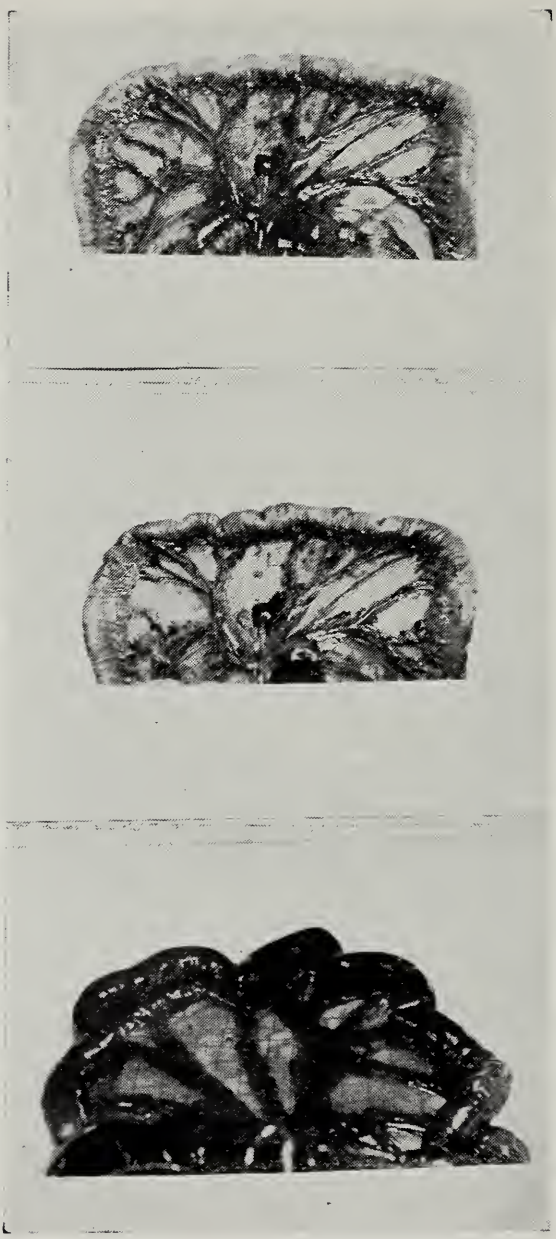


Fig. 10. Dog No. V.

Photograph No. 1 taken after tying the entire superior mesenteric artery.

Photograph No. 2 taken thirty minutes later.

Photograph No. 3 taken twenty-four hours later. Post mortem. Shows hemorrhagic infarct.

count most readily for the escape of the blood-making infarct. Also infection explains more readily the progressive character of the occlusion noted in many cases. Most cases met with clinically are associated with some infective condition. Those due to embolus may be from infected emboli. The remarkable case reported by Chiene, in which the celiac axis was entirely occluded and collateral circulation established, indicates the great possibilities of compensating circulation in this region.

The great size and extensive area supplied by the superior mesenteric artery, compared with the relatively small inferior mesenteric, makes easily accountable an infarct when the entire vessel is occluded. A sudden occlusion with the consequent phenomena of tonic intestinal contraction for some hours would make impossible a compensatory circulation before lethal changes in cells and vessels would take place and easy infection come from the intestinal tract. However, when we think of a branch or even several branches of the superior mesenteric itself being occluded, we wonder why there should ever be death of the part, considering the marvellous provision for collateral circulation through the numerous vessel loops in the mesentery. Such a free and large anastomosis should easily compensate for considerable occlusion. This beautiful arrangement of anastomosing vessels is provided in man and not in dogs!

What of diagnosis? A mortality of 94 per cent would indicate that diagnosis is most easily made post mortem. To make a reasonable tentative diagnosis would be admirable. To make a positive diagnosis would serve only to accent the egotism of the diagnostician. A picture of ileus is the most common. Sudden and intense abdominal pain, nausea, vomiting, collapse, subnormal temperature, rapid low tension

pulse, rigid abdomen and distended later, perhaps bloody stools if diarrhea occurs,—a picture like intestinal obstruction or intestinal perforation. The bloody stool is the most distinctive sign, but may be confused with intussusception. If given such a picture and an additional known source of embolus or a previous embolism elsewhere, we might guess a diagnosis.

The treatment of so grave a condition might be bettered by some forethought. A thorough knowledge of the anatomy and probable pathology in addition to extensive experience in abdominal surgery would be the most favorable equipment for one to have with which to meet such an emergency. A very few cases have been reported which have recovered as has been indicated. They come to us unexpectedly after a diagnosis of other trouble has been made. A resection is sometimes possible with a favorable result. Probably more often it would be wiser to simply fix the gangrenous bowel in the abdominal wound and drain as advised by Moynihan. There is no doubt that in many cases the process is progressive, and what appears at first to be a promising case disappoints because the infection and gangrene travel further.

The following two case reports are from my experience:

1. January 30, 1908. L. M., male, white, farmer, married, aged 26 years.

Family history.—A brother had an appendicitis; negative otherwise.

Pevious history.—Last October suffered five days from an attack like the present one.

Present trouble.—About 4:00 A. M. today, after getting up to make fires, he went back to bed and began to have severe pains in the abdomen, followed by vomiting. A physician arrived about 8:30 A. M. and made a diagnosis of acute appendicitis. Later in the day he was moved seven miles in a spring wagon to the train, and came nine miles on the railroad to

the hospital. He suffered great pain all day. Received castor oil, enemas, and morphine. I saw him later in the afternoon. He had a rigid abdomen; temperature, 97° F. I made a diagnosis of perforated appendix.

Operation, 8:30 P. M.—Found perforated appendix, and escaped fecal concretion; also a thrombosed mesentery in the lower part of ileum. Removed appendix; resected forty-five inches of ileum with end-to-end anastomosis. Drained. Wound partly closed. Shock. Proctoclysis. I saw the patient only once more, the following morning. Bedside notes indicated that the two days following operation temperature went to 99° F. He had much pain and considerable abdominal distention. Passed gas after enemas. After the second day his temperature went to 102° F. On the seventh day he had a large bowel movement, and on that day a bloody serum escaped from around the rectus muscle. Next day it had a fecal odor. No fecal discharge and the finger in the wound seems to find it walled off. A loop of dark bowel in the pocket. The last two days of his illness he suffered much more pain, and the abdomen became more distended, pulse weakening and more rapid, dying February 9, after living eleven days. No autopsy.

July 6, 1913. F. J., farmer, male, white, single, aged 21 years.

Family history.—Good.

Previous history.—Always had good health, a fine physique, no injuries.

Present trouble.—Tuesday, July 1, became sick after dinner. Went to his work, but had such severe abdominal pain had to quit and come home. Attributed the pain to indiscretion in eating at a party attended the night before. Called physician for relief that night. Doctor gave purgatives; and the bowels moved. Wednesday, 2d, had a chill. Thursday and Friday, 3d and 4th, still suffering, but was out of bed and going about some. Friday he ate some and had more pain, so went to bed again. Quite sick all day Saturday, 5th. I saw him first Sunday, 6th, 10:00 A. M., a robust, healthy specimen of manhood, lying in bed with his knees slightly flexed and without apparent suffering. Abdomen, tympanitic and tender all over; more tender to the left of the median line. Diagnosis, appendicitis and perforation. Removed to hospital, and operation 3:00 P. M. Found abdominal cavity full of bloody serum. All of the intestines discolored, and especially the jejunum. Looked im-

possible to resect. Selected the mesentery that was hard and thrombosed, and the bowel which was darkest, and did resect sixty inches. He died in five hours. No recovery from shock.

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DISCUSSION

Dr. A. A. Law (Minneapolis, Minn.): Dr. Nifong has spoken of the difficulty of diagnosing these cases of infarct. This was brought home to us so recently that I cannot refrain from reporting a case now fresh in my memory.

I was called to the country in consultation with two University graduates, both competent men. The case in question had been examined by a very competent medical man the day before. He had advised immediate operative interference. The only symptom the patient had had, covering a period of two weeks prior to the time I saw him, were rigors and high temperature. This temperature had been constant. His temperature at the time I saw him was 107.4°. He had given no symptoms of any kind,—nothing referable to his abdomen, no pain, no vomiting, nothing except chills and increased temperature.

When I saw him he had a slightly distended abdomen with a little fluid in the flanks. According to his history, his bowel movements had been normal, although test for occult blood had not been made. His leucocyte count was 23,000, and his p. m. n.'s were 90 per cent. This man was obviously not operable. We made a tentative diagnosis of septic thrombosis of the portal vein. He died 24 hours later and at the autopsy he was found to have had a septic thrombosis occluding his portal vein. His liver was soft and greatly enlarged. He had numerous infarcts in his mesentery and eight small areas of gangrene of the small bowel. In most of these cases I believe it is almost impossible to make a positive diagnosis.

Dr. Arthur E. Benjamin (Minneapolis, Minn.): I saw six years ago a case of a young man of twenty-five years who had been sick two days. I was called in the evening of the second day. He gave only symptoms of vomiting, and had no rise of temperature when I saw him, but his abdomen was somewhat distended. I immediately took him to the hospital, and operated on him, and found the whole of the jejunum black, that is, dark-colored, and a thrombosis or blocking of the mesenteric vessel, so that there was obviously nothing to do. We immediately closed the abdomen, and the man died. The bowel was absolutely dead,—as much as twenty feet of it,—so that we did not attempt to do anything, but simply let him alone.

Dr. Arthur E. Hertzler (Kansas City, Mo.): I would like to make just one suggestion as to the possible etiology of these cases. I have been trying to figure out some of the points in this problem, and it seems to me that the vessels are practically end arteries, because of the acute angle at which the anastomosis with each other takes place. For instance,

you have an arteria intesteni attenuis coming down this way (indicating) forming loops. Where the vessels form an acute angle in making the loop in this manner (indicating on blackboard), and you get virtually a valve so that in attempting to force the solution along here you get practically the same result as you do in the valves of a vein.

Where the vessels pass around the gut I rather suspect that the essayist's drawing was schematic. Because the vessels in going around the gut go around in a complete circle, there is little or no anastomosis between the various vessel loops that encircle the gut. So that, if one studies those things from that point of view, I think the problem of mesenteric thrombosis resolves itself down simply to a question of thrombosis as of any end artery.

Dr. Frederick A. Dunsmoor (Minneapolis, Minn.): I do not know that it is so uncommon for a patient to survive. I recall two cases where one patient died and one lived. I was called to Duluth Dec. 28, 1912, in consultation with one of our members, Dr. Stewart, and saw Mr. A., who had a plugged vessel, exactly as described by the essayist. We removed four feet of the terminal end of the ileum, which was gangrenous. I made a V-shaped incision of the mesentery and lock-stitched off the circulation at that point, and he immediately made a good recovery.

Another case soon after came to the St. Barnabas hospital. There had been a delay of about five days. There had been so much absorption of poisoned contents of bowel that although the same procedure was used death followed 20 hours afterward.

I believe it is not so much the absolute etiology of these cases as it is how long the condition has existed, so that the absorption of the decomposed contents of the intestine produces death, less that the condition of the mesentery before the operation is made.

Dr. Nifong (closing): I believe that it is not the anatomical factor that makes the difficulty in collateral circulation, but, rather, I think it is demonstrated that collateral circulation can be established, provided we do not have other factors, like infection.

I think infection is the greatest factor in producing the infarct and gangrene, because infection can come so readily from the long intestinal tract, and be so readily absorbed when there is any temporary stopping of the circulation. Without

nutrition lethal cell changes will take place and infection escape into this area in which the vital blood flow is occluded.

The great length, that is, the extensive area supplied by this one large vessel, is another factor in the matter. The superior mesentery takes from the duodenum clear down to the descending colon as its area of blood supply. If the entire vessel should be occluded you can readily see how much additional work would be suddenly put on a small vessel like the inferior mesentery from the vessels above. It would be impossible for this circulation to be established early enough to supply sufficient nutrition and prevent cell death and infection.

I do not see how there could be a finer arrangement for anastomosis than we see in these mesenteric vessels. There is nothing in the rest of the human anatomy that compares with it. We know that those cases recorded where extensive collateral circulation was established must have taken place very gradually and not have been accompanied by infection. Such is the case reported by Chieni.

OPERATION FOR PREVENTION OF RECURRENCE OF INTUSSUSCEPTION

John Prentiss Lord, M. D., F. A. C. S.

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My experience with intussusception has not led me afield from the accepted views held by those who have contributed to our recent extensive literature upon this subject. Epitomized, these are, that a mobile large cecum is the most frequent cause; that diarrhea and bloody stools are a result, and probably rarely a cause of intussusception; that cases treated early by the usual mechanical methods may succeed in a considerable proportion of the cases, but that persistence in these methods with the delay incident thereto furnishes the late hopeless cases brought to the door of surgery. The mortality from surgical intervention, therefore, is probably as high as from 66 to 90 per cent. Immediate or early surgical interference reduces the death-rate very greatly, but the cases come so persistently late to every operator that no one has been able to report a considerable number of consecutive early cases. The vast majority of patients are those in whom other measures have failed, the favorable period, from six, twelve, or twenty-four hours, having passed. The mortality from other acute abdominal conditions did not improve while medical treatment was adhered to.

Intussusception should be regarded as a surgical condition from the beginning, and surgery should be resorted to as soon as early mechanical measures, of

limited duration, have failed. In no case should the treatment delay the summons for surgery. In this way the great mortality will be reduced. Cases in which operation is done within twelve to twenty-four hours, are the ones most frequently successful. Earlier diagnoses should be made, and more prompt surgical intervention instituted. Fixation of the mobile cecum, appendectomy, and shortening of the ileocolic mesentery, to prevent recurrence, should be done, if the condition of the patient permits it, unless other etiological factors exist, in which case individual indications should be met. Infants bear fluid loss even less well than adults, and greater pains should be taken to restore fluids to the body by every avenue, and greater precautions should be observed because of this. The importance of this feature of treatment has been emphasized by all those who have written upon intestinal obstruction, more particularly Hartwell. This especially important indication should therefore be properly met in infants.

The use of aerohydrostatic pressure by means of an ordinary or a Davidson fountain-syringe, the latter with sufficient tubing to procure the exact amount of pressure tolerable, according to the age of the child, as recommended originally by Forest, is good practice, if used tentatively pending the arrival of the surgeon, to the end that no time be lost in makeshifts.

Kelly, in his "Surgery of Diseases of Children," I think logically summarizes his position as follows: "It is my opinion that modern results indicate the rule that no case at any age should be laparotomized without a correct application of aerohydrostatic method of reduction; and, if this fails, that no case at any age *but* should be subjected to laparotomy within the hour."

The following unique case may be of interest be-

cause the patient was operated on to prevent recurrence of intussusception. An infant of eight months had just been relieved of the third severe attack within five and one-half months. The symptoms were characteristic, and in the first two attacks relief had been obtained by hydrostatic pressure, inversion, and manual compression of the mass. The last attack failed to respond promptly to the usual treatment, and collapse and unconsciousness rapidly supervened. The child was brought one hundred and forty miles to the hospital by the family physician (Dr. C. C. Paxton), who by diligent effort, continued upon the train, had succeeded in relieving the conditions an hour before arrival.

The question arose as to what could be done to prevent a recurrence. As about 75 per cent of cases of intussusception occur at the ileocecum accompanied by a mobile cecum and ileum, with the appendix acting as the exciting cause, it was advised that the appendix be removed, and the cecum and ileum be firmly anchored by shortening their mesenteries. At operation the following day, a large, long, intensely injected appendix, lying alongside of an edematous, ecchymotic ileum and mesentery, the latter four or five inches in length, gave ample proof of the correctness of the etiological factors in the diagnosis. The appendix was removed, and the mesenteries of the cecum and ileum reefed with silk sutures. Recovery was complete; and there has been no return.

I find no record of an operation done *solely to prevent the recurrence* of intussusception, although fixation is frequently done at the time of operation for the relief of intussusception, in which the appendix and a long ileocecal mesentery have been the causative factors.

DISCUSSION

Dr. Frederick A. Dunsmoor (Minneapolis, Minn.): I am going to find fault with Dr. Lord for saying one thing in his paper, namely, that the mortality following a certain type of operation, *from the operation,* is 60 per cent. I do not want that to go out, because I do not think the mortality is from the operation at all. I do not believe such mortality comes from the operation, because we know that every single one of these cases will result in death unless operated upon; and it so happens that the report of the United States over, even from the best hospitals, is that obstruction of the bowel, taking all the groups together, no matter when operated on, results in death in about 50 per cent; that is to say, half of the cases which have been operated on for obstruction of the bowel have recovered.

That shows what an important subject the Doctor has taken up.

Two points which he mentioned we should think a lot of. The supply of fluid to and through the system should be inaugurated as rapidly as possible, and more especially with children, irrespective of where it is put into the system.

The second point is, that, when the obstruction is relieved, the intestine should be opened, if you are not sure it is going to empty itself, and the poisoned contents allowed to escape, and the intestine filled up with the normal salt solution at once, and the opening closed.

I believe that is a very important procedure in children.

I am going to mention the case of a physician who brought his child to me a distance of a hundred miles. The intussusception was relieved, and the child made a rapid recovery. Six months later he brought the same child in for obstruction of the bowels with adhesions. It made a good recovery.

About two months later the mother came in the hundred miles with this same child with a supposed obstruction. He had ordinary colic. The mother had been frightened. But they had come in at once with the first symptom each time.

I am certain the safest thing in all these cases is to operate the moment the diagnosis is made. I believe that in some cases it is thought that the obstruction has been relieved by enemata and the contents of the lower bowel evacuated when the reverse is true and fatalities have followed.

Dr. J. Frank Corbett (Minneapolis, Minn.): I would like to call attention to a phenomenon I have seen in an experi-

mental laboratory, as perhaps this has some bearing on the question of intussusception.

By occluding the mesenteric circulation of the bowel, both direct and collateral, to a small extent, we have been able to produce artificial intussusception. This would not occur where large segments were tied off, but it resulted from the complete occlusion of the blood supply to the small segments. This is corroborated clinically by the fact that thromboses are sometimes found in connection with intussusception.

Dr. St. Elmo M. Sala (Rock Island, Ill.): I think one important point in Dr. Lord's paper was this,—the removal of the appendix in all cases because I believe it has a good effect on the mortality. In the majority of these cases the appendix is involved, and it is under pressure, and the small appendiceal artery is occluded. A lot of these patients die from appendicitis after you have reduced the intussusception.

I am quite sure that the removal of the appendix in an eight-months old child in the last year saved the child. I believe I would have lost this patient from appendicitis after the operation if I had not removed it.

Dr. Van Buren Knott (Sioux City, Iowa): I do not believe mobile cecum, so called, is particularly to be held responsible for intussusception. In a reasonable experience with intussusception I have seen two cases in children in which the children had a complete pericolic membrane extending from the region of the hepatic flexure down to and beyond the cecum, to the point usually occupied by the bloodless fold of Treves, fixing the cecum absolutely and firmly to the lateral pelvic wall. Therefore, in these cases in particular mobility of the cecum could not be held responsible. Nor do I believe that operation to prevent recurrence of intussusception after the child has recovered from that particular attack, would be justifiable. I should prefer to wait until the child had a further attack of intussusception, and at that time, if necessary to operate, doing whatever seemed to be best to prevent recurrence.

Dr. William H. Magie (Duluth, Minn.): I was just going to say a word about appendicitis in connection with intussusception. I have had two cases of intussusception in which appendicitis was present at the time of operation, and in both of these cases the child had given a history of attacks of appendicitis before, in which they lasted a few days and subsided.

In these cases, of course, the appendix was removed, and found to contain pathologic material, namely, pus. They both recovered.

This led me to believe that in cases of intussusception it is our duty to remove the appendix, whether it shows signs of disease or not, for fear that it might at some future time cause another attack of intussusception.

Dr. J. Rilus Eastman (Indianapolis, Ind.): I would like to speak, briefly, of a case precisely like the two reported by Dr. Knott. Last Tuesday morning I operated on a child six months old for intussusception.

We found a condition like this (indicating with diagram on blackboard). We found the ileum passing up into the cecum and the tip of the appendix hanging out, red and inflamed. Here was a dense parietocolic membrane passing across the intussuscepted portion of the bowel,—a thick dense membrane.

I undertook to relieve the intussusception by making gentle traction upon the terminal ileum, seeking to express the ileum by pressure above, working down gradually upon it. I found that that was not possible until I had snipped off this very tightly fitting membrane, which passed across, as I indicate here, and fitting so closely over the intussuscepted segment of the bowel that it was at first difficult to discern it; but, passing the blunt point of the scissors under this band, it then became easily possible by pressure above, not so much by the traction upon the terminal ileum, to release the intussusception, after which, of course, we amputated the appendix.

It impressed me that the condition had come about because the terminal ileum with the valve of Bauhin, and the cecum had become tucked up under this constricting band, and that the constricting band militated against release of the intussusception.

Dr. Thomas C. Witherspoon (Butte, Mont.): Just a word as to the etiology of this trouble and a little procedure for its relief.

It seems to me that neither mobile cecum nor lengthened mesentery, as shown in the diagram, has anything to do with an intussusception.

In the normal peristaltic wave the segment of dilation precedes that of contraction, and allows the propulsion of the aliment onward. If there is a spastic contraction in the area of the ileocecal valve or a cicatricial or inflammatory stricturing, the oncoming contractual wave, not being provided for

by the compensating dilation at the valve, will drive that area on into the cecum, producing the intussusception, which then is carried on to the limit of mechanical possibility.

The shortening of the mesentery here (indicating on diagram) it seems to me is not a good surgical procedure. It is just that thing we get away from in our ordinary operations. It is something which may lead to further trouble, that is, to obstipated conditions in the bowel later. I have almost always done this.

Here in the diagram is the small bowel, and there we have the appendix. After taking off the appendix and tucking it in as we ordinarily do, I simply bring this small bowel (ileum) to the side of the cecum here until it passes this point of the appendix and suture the two together with catgut. This fixation disallows a reinvagination. I have never seen any reinvagination follow a procedure of the kind. I have always endeavored to relieve any constriction at the ileocecal valve.

Dr. Emil H. Beckman (Rochester, Minn.): I wish to report a case which I have never been able to explain, but which may possibly be explained by Dr. Corbett's remarks.

A man in our town went to work in the morning, and was immediately seized with violent paroxysmal pains, so that he had to be carried home. These pains continued most of the day with vomiting.

I saw him in consultation about five o'clock in the afternoon. The abdomen was somewhat tender, but not distended. There was no marked tenderness in any one particular locality, and there was no rise of temperature, and only a slight quickening of the pulse.

The man's physician thought he had appendicitis. I did not agree, but said that he must have some type of obstruction, to have such severe paroxysmal pain with vomiting.

He was operated on, and a rather harmless-looking appendix was found. Not being satisfied that this could have caused his violent paroxysmal pains, we began to go over the small intestine, and within about two feet of the beginning of the jejunum there were two places of intussusception, about six inches apart, one of them being reduced with considerable difficulty, and the other, about six inches higher up, being reduced very easily.

There was no growth in the small intestine, and there was nothing to explain the cause of the intussusception. The circulation, after putting on moist cloths, looked perfect; but

there might possibly have been an occlusion of some small vessel, as Dr. Corbett has suggested.

Dr. Malcolm L. Harris (Chicago, Ill.): I am sorry that I have to disagree with my friend, Dr. Lord, as to mobile cecum being such a factor in the production of intussusception as he has indicated. I have seen a number of cases of intussusception, but I do not think I ever saw one in which there was a movable cecum. The primary cause, and essential cause, in the production of intussusception, is something which causes an arrest of the peristaltic action of the bowel. That was demonstrated many years ago. If we go back over the experimental work done on the intestine we find that if you cause spasmodic contraction of a small area of the bowel, an intussusception may form at that point in a large percentage of cases. Just as one of the speakers said, if you will ligate or interrupt the circulation in the bowel in a small area—not in a large area, but in a small area—so that we have a narrow ring where there is an arrest of the peristalsis and a failure to dilate, an intussusception may result. That is a very simple and a very easy way to cause an intussusception of the bowel. As long as the bowel is freely movable, and peristalsis can move along it freely, you will never get an intussusception.

Intussusception was found to occur also in the early experiments in intestinal surgery, in suturing, where a foreign body was used which interrupted the passage of peristalsis. Intussusception frequently occured in the experiments; and tubes were used for the very purpose of preventing intussusception, until we had tubes six to eight inches long.

But what I am coming to is, that the primary cause in the production of intussusception is arrest in a very circumscribed area of the peristalsis of the bowel.

Dr. Byron B. Davis (Omaha, Neb.): I think that I can fully agree with Dr. Harris with reference to the etiology in almost all of these cases. I believe that nearly all of them are due to an arrest in peristalsis over a limited area; and if there is some definite thing which produces it and which is likely to reproduce it, it makes it very essential, when we are operating on these cases, to look carefully to see if we can find some mechanical reason for the production of the intussusception. I am led to say this by a recent case which I had.

In the diagram which I show you on the blackboard, let this be the large intestine, and here the small. Twelve inches

away from the ileocecal valve was a little "business" like this; in other words, we had a small, stubby Meckel's diverticulum, which remained when I got it out, and I found that was the cause or the beginning of the trouble.

A beautiful thing about this was, after reducing it, by using the tips of the gloved fingers, pressing here, making a slight dimple at this point, I could reproduce it. In a case like that, of course, if we do not do something at the time to prevent the reproduction of the trouble, we are going to have it occur again, and in this case, of course, it was very necessary to suture the diverticulum.

I think probably in a small percentage of the cases we have some mechanical reason for the production of the intussusception; but, I believe, in the majority of cases there is absolutely nothing we can do except to reduce the intussusception and then let it go.

But if we can find any special reason for its occurrence, as was found in this case, it seems to me at the time of the operation we should be on the alert to find it and do something to prevent its recurrence.

Dr. Harris: That interrupted the peristalsis?

Dr. Davis: Certainly, interrupted the peristalsis.

Dr. S. C. Beede (David City, Neb.): At the risk of making this discussion tedious I want again to call attention of this association to the plan of doing an appendicostomy in these cases.

In spite of the observations of three of the gentlemen who have discussed this paper, it certainly is a fact that the majority of intussusceptions are in cases where there is a mobile cecum. I am sorry that I cannot give you the literature, but two years ago I looked up the matter quite closely, and this was the result of the collected literature,—that in the majority of instances the cecum was at the apex of the invaginated bowel; and it was not the ileocecal valve at the apex, but the appendix that was at the apex.

I have not had a very wide experience in cases of invagination of the bowel; but I have had six or eight cases in children within the last three years, and in every one of these cases there was a mobile cecum and a tumor formed so that you could feel it through the abdominal wall at about the mid line.

If the appendix has anything to do with the production of

intussusception,—and I believe most of us have a sneaking notion that it has,—it is a good thing to remove it. If the cecum is mobile and it has anything to do with the production of intussusception, there is at least some reason for fixing it.

If it is desirable to empty the bowel of gas and to introduce fluids into it, the lumen of the appendix is a most convenient channel. Then, why not, in the ordinary case of cecal invagination, strip away the meso-appendix and pull that belligerent organ, and through a stab-wound in front of the iliac spine, fasten it there with a silk suture, snip it off flush with the skin and slip a catheter through its lumen into the cecum, and, if you will, on into the ileum? You thus fix the cecum securely, remove an exciting cause and provide safe bowel drainage.

Dr. Lord (closing): I am certainly grateful for the very generous discussion which I have elicited; and I will say that my conclusions were arrived at after a perusal of the literature, which seemed to bear out this hypothesis of mobile cecum. I had invariably found it in cases in which I had operated, and it had been my almost invariable custom to fix the cecum when removing the appendix, and to shorten the mesentery of the terminal ileum and cecum.

There has been no recurrence in any of these cases, and I am not so sure that there would not have been had I not taken this precaution. This procedure seems to be amply justified by the practice of others.

In regard to the point discussed by Dr. Dunsmoor in regard to the emptying of the intestines of their contents; I think that is a generally recognized principle that should be particularly observed in all cases of intestinal obstruction. This principle was enunciated some years ago, by Treves, I think, who immediately improved his mortality fifty per cent after resorting to this sort of procedure.

The position taken by Drs. Eastman and Knott,—that a fixed cecum in some of their cases was present and would therefore disprove the theory of the mobile cecum as a factor: I can readily conceive that a fixed cecum with a long mesentery on the terminal ileum would be an ideal condition favoring the development of intussusception.

The cases on which I have operated have been cases where the appendix seemed to be bound to the ileum so that the whole mass had gone into the cecum. The mechanical conditions as

observed, certainly gave the appearance of plausibility to this theory, because they seemed to have been a factor.

I agree that intussusception may be produced experimentally by interference with the circulation and by the application of irritants to the bowel, but it seems to me that, possibly an inflamed appendix, or one that was obstructed and that excited peristalsis within the organ itself, might transmit early to the ileum, contractions before the wave had passed on to the cecum, and that these conditions might favor the telescoping of the bowel.

I did not make these statistics. I cannot at this moment tell where I found them; but I did find the statistics that 75 per cent of the cases of intussusception are at the ileo-cecum and accompanied by a mobile cecum and terminal ileum; all of my cases have been of this type.

I have been criticised for operating on this case. I wish to remind you that this case was one hundred and forty miles distant from Omaha, that within a comparatively short time there were three alarming attacks, and it was only by the most prompt attention that the case was relieved. These people lived on a branch line of railroad. They rushed to the main-line train by automobile, when they found that the conditions were so alarming, with the child in an unconscious condition, and not relieved as previously. The question was, should I send these people home for this thing to be repeated, or should I do the thing that seemed to be the logical thing to do, put the viscera at this point in a condition to make it physically impossible for the trouble to recur.

THE DIFFERENTIAL DIAGNOSIS BETWEEN THE ACUTE PATHOLOGICAL PERFORATIONS WITHIN THE ABDOMEN, WITH SPECIAL REFERENCE TO DUODENAL PERFORATIONS—THE INDICATED TECHNIC OF OPERATION

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In a discussion of papers upon appendicitis read by Drs. Maclaren and Moore, at the last meeting of the American Medical Association, Dr. J. M. T. Finney, of Baltimore, said: "In appendicitis, in my judgment, the mortality is absolutely preventable. When a death occurs from appendicitis, it is prima facie evidence that somebody has made a mistake." My own experience teaches me that this position, with rational qualifications, is the correct one, and that the same statement holds true regarding peritonitis following all forms of pathological perforations within the abdomen, with one exception, that of typhoid fever, when the mind and sensibilities are obtunded. It is always possible, after operation, when the patient's equilibrium and that of his family and friends are restored, to get a history of antecedent symptoms in every case of acute perforation of the abdomen, whether the lesion be of the stomach, duodenum, gall-bladder, pancreas, or appendix—all of these in the male. In the female may be added infections or impregnation in the fallopian tubes. As Moynihan says: "Late

symptoms are too often the heralds of death; inaugural symptoms may be the cry for timely surgical assistance." Surgeons are all familiar with the classical symptoms of acute abdominal perforation, i. e., peritonitis, soon becoming general, following a sudden onset of severe, *excruciating* pain, short, quick breathing, an anxious expression of face, rigidity of the abdominal muscles.

The accurate differentiation between the different lesions depends, to a very great degree, upon the anamnesis. One must know the *initial site* of severe pain, since it becomes general later, and there may be no point of *greatest intensity*. If the history gives the usual signs of chronic gastric or duodenal ulcer, there is, as a rule, no difficulty in locating the perforation; however, a patient frequently remembers only a "slight indigestion," or no symptoms whatever, until the perforation took place. Careful questioning often proves contradictory to these statements, giving an entirely different history. When the history is unreliable, one must depend upon the physical examination. Alexander Miles differentiates between gastric and duodenal ulcer perforations as follows: "When the most tender area is in the left hypochondrium, the perforation has almost invariably been towards the cardiac end of the stomach; and when in the right hypochondrium, near the pyloric end or in the duodenum." Miles finds this association so constant that he relies upon it as a guide to the site of the incision. The physical symptoms of *duodenal perforation* and *appendicitis* are often so similar that it is difficult to distinguish between the two. Both are characterized by pain in the right iliac fossa; however, in duodenal perforation there is always pain in the right hypochondrium, which occurs only *exceptionally* in appendicitis. On the other hand, the intensity of the pain is not so great in the

latter as in the former, nor are the abdominal muscles so extremely rigid; consequently, in appendicitis breathing is not so short as in case of duodenal perforation. Here again, if these physical differences cannot be distinguished, one must rely chiefly upon the anamnesis. E. Stanmore Bishop (*British Medical Journal,* May 4, 1912) does not agree with Moynihan (*British Medical Journal,* February 17, 1912), as to the difficulty in diagnosis of cases presenting symptoms of gastroduodenal ulcer, but in which the origin of the trouble is elsewhere. Mr. Bishop says, "It is in the *differences* noticeable between the symptoms present in previous attacks that some assistance to a diagnosis may be found." He thinks these cases are very likely to be appendicitis of the "relapsing variety," and tells of instances in which the symptoms of earlier attacks were exactly those of gastroduodenal ulcer, which *must* have been the pre-operative *diagnosis* in the earlier years of their history. Later, *characteristic* symptoms of appendicitis developed.

Occasionally an acute perforation in the gall-bladder will present symptoms so similar to those of duodenal perforation that an accurate diagnosis may be made only by obtaining a careful history. Moynihan, in his book on "Duodenal Ulcer," tells of only one instance of acute perforation of the gall-bladder, in which the anamnesis revealed symptoms mimicking those of duodenal perforation. Since the patient seemed less dangerously ill than he would have been in the usual duodenal perforation, the case was rightly diagnosed as an acute perforation of the gall-bladder. On the other hand, a patient suffering from an acute perforation of the gall-bladder, operated upon by me, presented symptoms as intensely acute as I have ever seen in perforation of the duodenum.

It may be difficult to distinguish between acute

pancreatitis and perforation of the duodenum. A careful history in a case of acute pancreatitis will often show dyspeptic symptoms identical with those of gall-stones, while in duodenal perforation a history of previous indigestion is unusual, except that so classically described by Moynihan as hyperchlorhydria, which he calls duodenal ulcer. Mr. D'Arcy Power (*London Lancet,* July 13, 1912) says, "Indeed the absence of a history of dyspepsia, with the signs of sudden perforation * * * inclines one at once towards an exploration of the duodenum." Vomiting is also an important symptom pointing to pancreatitis. It must be emphasized that in an acute pancreatitis the pain is very severe, and the tenderness is in the pancreatic region. In four cases of acute pancreatitis operated on by the writer, there was a history of gall-stones, and gall-stones present, in three (two were hemorrhagic), and in the fourth (acute abscess) drainage of the gall-bladder was finally necessary to cure a second attack.

A variety of conditions may bring about acute perforation elsewhere in the abdomen, e. g., in the jejunum, which is almost invariably caused by ulceration secondary to the performance of gastro-enterostomy. I have never seen one. In the ileum or in the colon, the typhoid perforation occurs most often, and is the most important. Its location is usually in the ileum near the cecum, where its physical symptoms would be very similar to those of appendicitis, which may be a complication of typhoid; however, in a patient suffering from typhoid a typhoid perforation may usually be differentiated by the onset of pain (more sudden than in appendicitis), by increased vomiting, possibly a chill in connection with the pain, and by the accelerated pulse-rate. In typhoid, diarrhea is the rule; in appendicitis it is constipation. Practically the only source of assistance in distinguishing between a

typhoid perforation and gastroduodenal lesions (also possible complications of typhoid) is found in the antecedent symptoms. When an acute affection of the gall-bladder complicates typhoid, the occurrence of jaundice will be of diagnostic value.

Dr. George Armstrong, in a paper on typhoid perforations, read in London at the last Clinical Congress of Surgeons, differs very much from the usual text-book descriptions or clinical analyses of typhoid perforations. In his analysis, based upon a study of 15,224 cases of typhoid fever, there were 544 perforations, or about *one* in thirty and one-half; 83 of these occurred in the Royal Victoria Hospital, and 110 in the Montreal General Hospital. As he is a surgeon to these hospitals, one must realize that Dr. Armstrong's opinions have a sound foundation. He states that the first indication that perforation has occurred, is *usually* pain. The pain is often not severe in character. Dr. Armstrong emphasizes the fact that in the training of a typhoid nurse, she should be taught to send for a physician whenever a typhoid patient complains of abdominal pain.

The *second* symptom which he emphasizes as of greatest importance, is the change in facial expression. It is usually, in the majority of cases, quite evident. There may be pallor, a look of distress, restlessness, vomiting, chills, profuse sweating, general malaise, an unaccountable change for the worse in the general condition, a feeling among those in attendance that something has happened for which they cannot account. Tenderness and rigidity were present in some degree in about 85 per cent of all cases, and usually were present within the first two hours following the initial pain. Personally, I have always found liver dullness unreliable as clinical evidence in any form of abdominal perforation. Dr. Armstrong observed

that there was seldom a fall in temperature, and *always* a rise in the pulse-rate.

One must not forget in differentiating between acute lesions within the abdomen, that identical symptoms may have their origin in some intrathoracic lesion. They are usually the symptoms of appendicitis. Moynihan, in his book on "Duodenal Ulcer," quotes the late Doctor Maurice Richardson in this connection, as follows: "The diagnosis between acute thoracic and acute abdominal disease is always easy as soon as the characteristic signs of either are apparent. The chief difficulty in making a distinction is to recognize that the necessity for that distinction exists, for the thoracic symptoms are always masked by the more conspicuous and distressing abdominal ones. Once the attention is drawn to the possibility of a thoracic cause, not only for the thoracic, but for the abdominal, symptoms, an accurate diagnosis is perfectly easy." Regarding this quotation, I feel like repeating what Moynihan said to me not long ago, that Maurice Richardson expressed himself better than any other American with whose writings he was familiar.

In the female the diagnosis of acute conditions of the fallopian tubes and the ovaries, which have caused peritonitis, may be greatly aided by careful vaginal examination, in addition to abdominal examination. The only other diagnostic means is a minute inquiry into the previous history. However, if one has not been able to distinguish accurately between these conditions and a possible appendicitis or other intestinal lesion, either one may be easily reached through the same incision in the right iliac fossa.

All reference to blood-examinations in differentiating the lumen of the bowel so as to obstruct it, the performance of gastro-enterostomy would only increase the time required for the operation unnecessa-

rily. This advice applies to patients in a weakened serious condition. They are *late* operations. In *timely* operations with patients in *good* condition, the ulcer should be infolded, if necessary, practically obliterating the duodenum. This is done always *after* the gastrojejunostomy. Obliteration of the duodenum, whether by infolding or ligature suture, whatever the material used, should be regarded as a temporizing measure, giving time for the ulcer to heal. In two cases, on later occasions, we were able to demonstrate that the lumen of the duodenum was completely restored after a heavy linen ligature had been used—the linen had disappeared. Let me note also in corroboration of this principle, that I once ligated the fallopian tubes with silk at a Cesarean section, and within a few months the woman was pregnant again. Although I have had no experience with autogenous suture material for the obliteration of the pylorus, I am skeptical about the permanency of the results of its use. Resection of the duodenum is the only positive means of its permanent obliteration.

Drainage, local, pelvic, or both, should be employed as the judgment of the operator determines.

For purposes of illustration I have selected from my personal experience notes upon five cases of acute perforation of the duodenum, as they cover the field of this lesion as we meet with it in actual practice.

Case 1.—A man aged about thirty-three, was first seen on the morning of January 20, 1900. He had that morning been seized with very acute pain in the upper abdomen. The history elicited was that he had, from time to time, been a sufferer from "acid dyspepsia," for the relief of which he had been in the habit of taking baking-soda. The symptoms so simulated gall-stone colic that I made this diagnosis, and sought to reduce the pain by hypodermics of morphine, and the local application of heat. Late in the day I became suspicious that a wrong diagnosis had been made, and Dr. W. O. Bridges saw the patient with me, in consultation. We sus-

pected the possibility of a perforation, either of the stomach or duodenum, but were uncertain. The patient rapidly became very ill, and died the following morning, twenty-four hours after the first seizure of pain. A post-mortem examination disclosed an acute peritonitis caused by a perforation of a duodenal ulcer.

Case 2.—A taxidermist, aged thirty-five, curiously enough had a history as to acidity similar to that of Case 1. He, too, had taken baking-soda to overcome the acidity. This man was seized with a very acute pain in the abdomen at four o'clock in the afternoon. He consulted a physician, who gave him some morphine powders to control his "colic." Not receiving sufficient relief, he proceeded to take a number of drinks of whisky. The pain continuing, he went to the Clarkson Hospital at 10 p. m. He was seen by my assistant, Dr. Scott, who made a diagnosis of acute pathological perforation in the upper abdomen. I operated within the hour, and found a large perforating ulcer of the duodenum, which was closed by suture. Through a suprapubic incision a considerable quantity of strong whisky-smelling fluid was sponged out of the abdomen, and a rubber drain introduced. Convalescence was uneventful.

Case 3.—A man, thirty-three years of age, was taken with the symptoms of acute perforation of the abdomen. He gave a history of indigestion and hyperchlorhydria. He had had several attacks of pain in the upper abdomen, usually in winter time, the history of the attacks dating back eight years. The first site of pain and the tenderness were in the right upper abdomen. His physician's diagnosis was acute appendicitis. To my mind, however, the symptoms were positive of perforation of a duodenal ulcer. The operation was done at the Clarkson Hospital, twelve hours after the beginning of acute symptoms. Findings: perforation of a duodenal ulcer and local peritonitis. The perforation was sutured, practically occluding the duodenum; a posterior gastro-enterostomy was done. Recovery.

Case 4.—A man, twenty-eight years of age, gave a history of hyperchlorhydria and "hunger pain," dating back five years. After two days' prodromal symptoms, the symptoms of acute perforation developed. His physician's diagnosis was acute perforative appendicitis. The operation at the Clarkson Hospital, six hours after the first acute symptoms, disclosed a perforating ulcer of the duodenum, which was infolded, prac-

tically occluding the duodenum close to the pylorus. A posterior gastro-enterostomy was done, and a suprapubic drain was introduced. There was a prompt, uncomplicated recovery. One year later I successfully operated on this man for an acute suppurative cholecystitis.

Case 5.—A man, forty years of age, a dentist, of Harlan, Iowa, had suffered from numerous attacks of sharp pain in the abdomen. The last attack, from which the patient was in bed but convalescent, had continued for two weeks, and had been diagnosed as a cholecystitis. I was called to operate for *cholecystitis.* An incision disclosed a normal gall-bladder, and while I was examining this a perforation of the duodenum, with commencing leakage (bubbling of gas and then escaping chyme) was observed. This perforation, with the leakage, took place under my eyes while I was inspecting the gall-bladder, and for this reason is particularly interesting. The operation consisted of infolding of the ulcer, and the doing of a posterior gastro-enterostomy. The recovery was uncomplicated.

DISCUSSION

Dr. W. F. Reynolds (Kansas City, Mo.): Dr. Summers' paper is an exceedingly good one; and the suggestion in it in regard to bowel perforations is especially good.

The vast majority of perforated ulcers occur in the upper abdomen, the same as duodenal ulcers or gastric ulcers. They have a pretty definite symptomatology, existing prior to perforation; but there are cases in which the history is so meager and vague that the perforation comes on as a surprise. An experience I had very recently illustrates the point.

I was called to Arkansas to see a man who had had a terrific pain in the upper abdomen. He had given no history of trouble previous to this, not even of a mild dyspepsia. The doctor in charge of this man had fortunately taken all food away from him, and had kept everything from his stomach. Upon opening the upper abdomen we found a perforated ulcer at the duodenal-jejunal junction, a place where I think they seldom occur.

Another man worked all day at the side of a barber's chair, with no previous history of anything but a mild dyspepsia. He had an agonizing pain, occurring on a street-car when he was on his way home. The pain was so intense that he got off the car, and wallowed around in the street. The car was stopped, and he was carried into a drug-store.

This man had a perforation of the mid-ileum. He had had typhoid fever sixteen years previously, but no history of recent trouble.

The cashier of the Smith-Townsend-McCord Dry Goods Company worked at his desk until 11 o'clock, when he was seized with an agonizing pain in the upper abdomen. He was taken home. I operated upon him six hours later. He had a perforation of the anterior wall of the stomach, without previous history of trouble.

Dr. August F. Jonas (Omaha, Neb.): I would like to ask a question of Dr. Summers. Is it not a fact that almost all of these perforations are associated with intense rigidity in the abdominal wall?

In the majority of the perforations I have seen, immediately after the perforation there is intense rigidity, which passes away after a few hours.

It seems to me that is a very important symptom in connection with the perforation of the abdominal viscera anywhere.

Dr. Summers (closing): In my paper I spoke first of pain and then of rigidity. Always, I think, without exception, I have had numerous cases of traumatic perforations, and I have never yet seen a case without there was muscular rigidity. It is always my index to interference.

I think pelvic drainage in many of these cases is very important. I believe the elevated position of the trunk, the so-called Fowler position, is valuable. That has been my practice in the comparatively small number of cases I have had,—eight or ten acute perforations of the duodenum.

EXCESSIVE MORTALITY OF HIGH INTESTINAL OBSTRUCTION, WITH A REPORT OF A CASE

William W. Grant, M. D., F. A. C. S.

DENVER, COLORADO

There is probably no field of surgical research at the present time that is of more interest and importance than the cause of the excessive mortality in high obstructions of the small intestine. Until the last year or two not even a plausible explanation had been offered. While the death-rate is high in any part of the small intestines when obstruction is complete, yet it has long been recognized to be greater when the seat of obstruction is in the upper jejunal and duodenal tract. While due consideration has been given to the prior investigations of Murphy and Vincent *(Boston Medical Journal,* 1911, ccxv, 684), of Roger, and a few others, it is perhaps only necessary on this occasion to refer to the more recent investigations of Whipple, Stone, and Burnheim, of Baltimore *(Journal of Experimental Medicine,* 1913, xvii, No. 3, 286, and other publications); and of Hartwell, Hoguet, and Beekman, of New York, and the recent work and articles by Draper *(Jour. of the A. M. A.,* 1911, vol. 57, 1338, and September 26, 1914).

These investigations have given the chief important bibliography and data to the present time. The experiments have been made mostly on dogs, the details of which it is needless to attempt to give on this occa-

sion. The conclusions of Whipple, Stone, and Burnheim are, that death is due primarily and chiefly to a poison secreted or produced by the duodenal mucosa and stagnant contents above the seat of obstruction, and that the loss of water from the tissues into the paralyzed distended intestine and stomach is a secondary phenomenon.

Hartwell, Hoguet, and Beekman conclude that death is due, first, to the excessive water loss which regurgitates into the stomach and is vomited; and, secondly, to a toxin which is the result of bacterial invasion of the paralyzed and traumatized intestinal wall. Both schools, New York and Baltimore, agree that general bacteremia, though plausible, is not tenable. These investigations seem conclusive in many respects, but it probably cannot yet be affirmed that the cause of death is fully and finally determined; yet the future seems bright and more promising of good results, which is the aim of all investigations. The supreme test is the final application of all animal experimentation and laboratory results to the bedside. In the last year two typical patients with mechanical obstruction have been operated upon and treated by me. The histories are interesting, and may be regarded as instructive, even in the absence of satisfactory results.

Case 1.—H. F. R., male, aged 51 years, grocery merchant in Iowa. He came to Colorado seven years ago for asthma, bought a ranch, and engaged in the cattle business. He recovered early from asthma, and was in good health until his fatal illness, which commenced August 15, 1913, at which time he took an active cathartic. The effect was severe griping pains in the abdomen with satisfactory result. Next day, on account of pain, he sent for a physician, who proceeded to give cathartics, which were vomited, and rectal enemas, which were of no avail, and hypodermic injections of morphine to relieve pain. No other measures were used, and the vomiting continued and finally became fecal. On August 22 he was taken to St. Luke's Hospital in my charge. On admission his

temperature was 97.4°; pulse, 92; restless, not speaking above a whisper without great effort. With a full week of vomiting and no nourishment his exhaustion was not surprising, and yet the pulse was fair in volume and strength. There was not much tympanites or pain on pressure, but a globular tumor around the umbilicus. The abdomen was flat and dull below. Obstruction was manifest, and I operated immediately. Subdermal salt solution was given on the operating-table, and ether for anesthesia. The operation revealed the upper small intestines lying in parallel rows and enormously distended, and the lower intestines collapsed. There was a volvulus of the jejunum with localized gangrene at the seat of obstruction. No general peritonitis existed, and adhesions were limited to the loop at the point of obstruction. No perforation and no free fluid in the peritoneal cavity. The intestines were easily withdrawn, and the partially gangrenous loop ready to rupture was incised, but not divided, and a rubber tube put in each end and fixed. By gentle manipulation the intestines were evacuated, and then irrigated through the artificial opening with warm salt solution, every care, of course, being used throughout the operation to prevent soiling of the peritoneal cavity. The intestine was stitched to the peritoneal incision, and the gut protected by a wire screen over moist sterile gauze. Stomach lavage was used before the patient left the operating-table; appendix, gall-bladder and stomach were examined, and presented no evidence of perforation or disease. Vomiting ceased immediately, and his general condition seemed very good. On August 23, 8 A. M. (second day) his temperature was 98.5°, and his pulse 94. At 8 P. M., the temperature was 99.5°, and the pulse 102, and respiration 20. August 24, same. August 25, 8 A. M., 98.5°; 4 P. M., 101.5°; pulse, 116. August 26, same, morning and evening. August 27, 8 A. M., 100.5°; pulse, 126. August 28, temperature, 99° to 100.5°; pulse, 120 to 130; respiration, 14. August 29, temperature 101°; pulse, 120 to 130, intermittent and weak; respiration, 8 to 26. August 30, temperature and pulse fluctuated in same way. Early on the morning of August 31, the patient died as one in profound exhaustion from starvation, and yet from the very day of operation, on the 22d, he was given and retained a large quantity of food by stomach and rectum, and salt solution daily for hours per rectum, and at intervals peptonoids by rectum, as well as by the artificial opening after irrigation of the intestinal loops. At the end of the second day there was free and frequent fecal discharge

per the enterostomy wound, requiring frequent change of dressings. He seemed to improve for three or four days, and then relapsed into the same restless, exhausted state, and sleeping but little, yet free from pain, and with no complaint except weakness; no delirium and no mental confusion, but apathetic. No peritonitis developed. The abdomen remained flat, and presenting no ordinary signs of sepsis. It is surprising that with complete obstruction he lived so long without relief and without nourishment before operation.

Case 2.—This patient was a fine, intelligent boy, aged 10 years, living with his parents on a farm near Stratton, Colo. On September 2, while riding a bicycle alone in the country, his machine tumbled, and he was thrown on his left side, bruising, but not cutting, the left epigastrium, probably falling on the handle-bar of his bicycle. He reported suffering severe pain, but after a little while started home pushing, not riding, his machine two miles. On the second day he was visited by Dr. Beachly, of Stratton, who recognized the surgical nature of his condition, and brought him to St. Luke's Hospital, Denver, wiring me to be prepared to receive him. There had been no bowel movement, no escape of gas, and twenty-four hours after injury rather frequent vomiting. He arrived at the hospital September 5, with temperature, 102°; pulse, 116, the abdomen considerably distended, and painful on pressure. The stomach was greatly distended, and he was vomiting a great deal of liquid bile-stained material.

I operated immediately, making the incision to the outer side of the left rectus and extending the incision, as in preceding case, above the level of the umbilicus. The moment the peritoneum was incised a large quantity of dark liquid blood escaped from the peritoneal cavity. Examination revealed a kink of the jejunum. There were no adhesions of consequence. The obstruction was readily corrected; but deep in the belly, in the upper part of the jejunum, I discovered an abraded spot, at least an inch long, in the mesentery, with hemorrhage beneath the adjoining peritoneal coat of the intestine for distance of two or three inches. I closed the rent in the mesentery with catgut. There was undoubtedly a slow hemorrhage from this spot with considerable effusion of serum. There was no other lesion in the abdomen. There was no gangrene, and the color of the intestines below the obstruction rapidly improved under hot cloths and salt solution. To stop the regurgitation into the stomach and to drain the upper

loop of intestine, an immediate enterostomy was performed, and the intestines irrigated with warm salt solution. For two days vomiting was frequent, necessitating frequent stomach lavage. It ceased at this stage, and he took nourishment freely by stomach, with slow salt solution by rectum, with hypodermoclysis also. During the first two days he was at times quite delirious. On the second day, September 6, the highest temperature was 101.5°, evening, 99.5°; morning pulse, 110. September 7, temperature, A. M., 98.75°; P. M., 99.75°; pulse, 108. September 7, 8, and 9, they fluctuated in the same way; no delirium, and the patient seemed better, though rapid emaciation was manifest in spite of the exhibition and retention of a large quantity of food by stomach, rectum, and small intestine through drainage-tube. On September 10 and 11, the morning temperature was 100.2°, and evening 99° and 98.5°, reversing the usual order. September 12, 6 A. M., temperature, 98.5°; evening, 99.75°; pulse, 116. Patient weaker and again delirious by spells. September 13, temperature the same; pulse, 140; and respiration, 20, never much accelerated. From this time on, exhaustion and emaciation were rapid, with same moderate temperature, but fast pulse, until his death on September 19, just two weeks from the date of operation, and twenty days from the date of injury.

The fecal discharge around the intestinal tube soiled the wound, greatly inflamed the skin over the abdomen, and prevented union of the wound around the tube. This condition was doubtless due to the pancreatic secretions. This process was manifested on the fifth day after operation, and as vomiting had ceased, and he was retaining food by both stomach and rectum, I closed the intestinal stoma under local anesthesia, and applied sterile vaseline to the surface of the abdomen. He progressed very well for a day. There was no fecal discharge by rectum, even with enemas. Vomiting recurred, and I found it necessary to do a second enterostomy, which was performed at the upper angle of the wound under local anesthesia, and above the previous intestinal opening. A large catheter was used for drainage, and the intestines irrigated through it also. Vomiting ceased almost entirely. Nourishment was retained, but in spite of every care and attention, the little patient went on from bad to worse until the end on September 18.

There was no general peritonitis, no distention of consequence, and no pain except from the irritation to the outer surface, due to pancreatic juices chiefly, no general sepsis ex-

isted. In low obstruction the toxin, it is claimed, is not so virulent. In the experiments on dogs death occurred usually in three or four days from complete obstruction.

The treatment, so far, is, in the main, unsatisfactory because unsuccessful. In the condition of complete paralysis existing in these cases, after three days of complete obstruction, enterostomy seems a necessity, even with frequent stomach lavage. I used eserin and pituitrin to stimulate intestinal mobility, but with unsatisfactory results. Vaccines made from the duodenal and jejunal mucosa of healthy animals have been suggested. Draper (Maury) found from experiments that dogs fed with such substances lived longer than the uncontrols. Hartwell and Hoguet admit that while the drainage from the tissues can be replaced by salt solution, it is not sufficient to prevent death. It was abundantly supplied in both my cases, and kidneys acted well in both. Whether the tissue drainage into the distended intestine and stomach is primary and the toxin a secondary evolution, or vice versa, makes little or no difference with the treatment. We know that the former can be successfully met, but the toxin, the production and nature of which is not yet understood, destroys the patient very much as would be expected in a steady process of exhaustion from starvation.

Whether enterostomy should be performed in very recent cases brought promptly to operation for mechanical ileus with no serious intestinal lesion or preexisting disease, may be a debatable question. But after twenty-four hours, with intestine and blood-vessels distended and paralyzed, and vomiting frequent, there can be no doubt that enterostomy gives more prompt relief to symptoms, and prolongs life, at least. Until some measure of relief is found for the toxin, the result of the obstruction, the mortality will continue high.

It is to be hoped that by reporting such cases we may promote interest and inquiry in the solution of a grave and important class of cases.

DISCUSSION

Dr. Amos W. Abbott (Minneapolis, Minn.): I was very much interested in the Doctor's paper, for several reasons, among which is the fact that I have had cases of this kind which went wrong. There is probably no one in this room but has had more or less experience with this difficulty. I want to recite, briefly, a case for Dr. Grant's encouragement, which I saw but a short time ago.

The woman, fifty-nine years of age, was suddenly taken with pain in the abdomen, having a temperature of one hundred and four. I could get no more definite history. Finally, after four days of suffering, she was referred to me. In this time she had been continuously vomiting. There had been no movement of the bowels and no passage of gas.

As soon as she could be gotten to the hospital we opened the abdomen, and found an obstruction, I should think, perhaps a third of the way down the ileum. The ileum was attached by an adhesion in the pelvis.

The curious feature of the trouble was, that there was a distention of the entire intestine, not only above but below the obstruction. There was no peristalsis whatever. We could induce no peristalsis in any way. The bowel was not black, but was a very dark-brown, and on the surface there were frequent spots of ecchymosis.

The woman was in a desperate condition, and I did not expect her to live. However, as I said, we opened the abdomen and found this condition. The tension was so great that it was necessary to take out the entire abdominal contents; and then we perforated the intestine, put it in a glass tube, and washed it out thoroughly above and below. It then resumed a perfectly ribbon-like, flaccid condition. There was still no peristalsis in the bowel whatever—no attempt at any contraction, as far as we could see.

Partly because I had done it one one or two occasions before in extensive peritonitis, I poured a little ether into the abdomen, and immediately there was a little contraction. I then poured in a whole half can of ether, and let it boil out, closed the intestinal wound, and then made several drainages from different points through the flanks and Douglas' sac.

The woman made a good recovery, so that I feel hopeful Dr. Grant will be more fortunate in his next case.

Dr. John P. Lord (Omaha, Neb.): The members will perhaps recall that I talked on the subject of enterostomy some years ago, and took some considerable pains to develop the subject.

It is my experience that an enterostomy of the jejunum is a rather dangerous procedure. This portion of the gut is so small when collapsed that almost any tube which you may introduce by this method, stands a very good chance of practically obstructing the lumen; and I think it behooves everybody who uses this particular method to use a very small tube, and to be very particular not to include too much of the intestine, in the purse-string sutures in these particular cases.

I am not raising this point to criticize in any way or to take exception to Dr. Grant's report. I believe, however, that enterostomy in the jejunum is beset with especial disadvantages.

Dr. Grant (closing): Dr. Abbott's case, as he said, was in the lower intestine, and the experiments prove that operations for obstructions in the lower intestine are much more successful than operations of any kind in complete obstruction of the upper intestine.

It is important to operate on these cases in the shortest possible time. Of course, if it is done within twenty-four hours the chances are that in some cases enterostomy may not be even necessary, but in most cases we do not see them very promptly.

In one of my cases, as I stated, it was seven or eight days, and in the other at least three days and nights, before the patient came to the operating-table.

The surprising thing is, that, after operations, these cases seem to do well for a short time. It has been proven, I think, satisfactorily, that the fact of this tremendous loss of water from the tissues is not of itself sufficient to cause death. The patients die just the same.

The food seems fairly well digested when it is given, as it can be, through the small intestinal loop. If you use a rubber tube, and do not put it far into the loop, there is no danger of the obstruction of which Dr. Lord speaks.

It is probably very well established today that glass drainage-tubes are the most thoroughly aseptic, and can be kept in this condition better than any other; but in the intestine, always infected with bacteria, the rubber drainage-tube answers our pur-

pose very well indeed, for it is in use only a short time, as a rule.

The toxin—not a general sepsis, but the toxin which is absorbed from the obstructed loop of the upper intestine—is generally believed now to be the poisonous agent that produces the fatal result.

So far we do not know exactly what that is. The more recent experiments show that it is not bacterial, but that it is a toxin, and that with the tremendous loss of water produces the high mortality.

SUBSTITUTE OPERATIONS FOR THE MORE RADICAL SURGICAL WORK ADVISED BY MR. LANE IN INTESTINAL STASIS

Arthur E. Benjamin, M. D.

MINNEAPOLIS, MINNESOTA

Patients suffering with intestinal stasis are generally very annoying to physicians because of our acknowledged inability to cope with the manifold symptoms of the disease, and because of the present uncertainty of the best mode of treatment.

The symptoms of the disease now recognized as intestinal stasis, have been misinterpreted and often characterized, in the past, as stomach trouble, liver complaint, sick headache, dyspepsia, biliousness, hyperacidity, nervous dyspepsia, neurasthenia, etc. All or some of these symptoms may have been present, but these terms do not as thoroughly cover the phenomena present as the term *intestinal stasis.* It at once suggests to the mind some underlying factor responsible for the condition, and significant reasons for the multiplicity of the symptoms complained of.

Unquestionably, intestinal stasis may occur in children; but its causes in later life are often due to habits of indiscretion in diet and to unhygienic modes of living founded in childhood. In a child who is proportionately well developed and who is given pure suitable food, with all parts of the body kept free of infection,—such as the teeth, tonsils, respiratory

apparatus, sinuses, etc.,—and active daily general exercise kept up, the muscles of the body, including the abdominal and the intestinal, will keep up the required tone so as to take care of the digestion and elimination of waste products. No overloading of any portion of the colon nor sagging is likely to occur, nor will stasis develop during life.

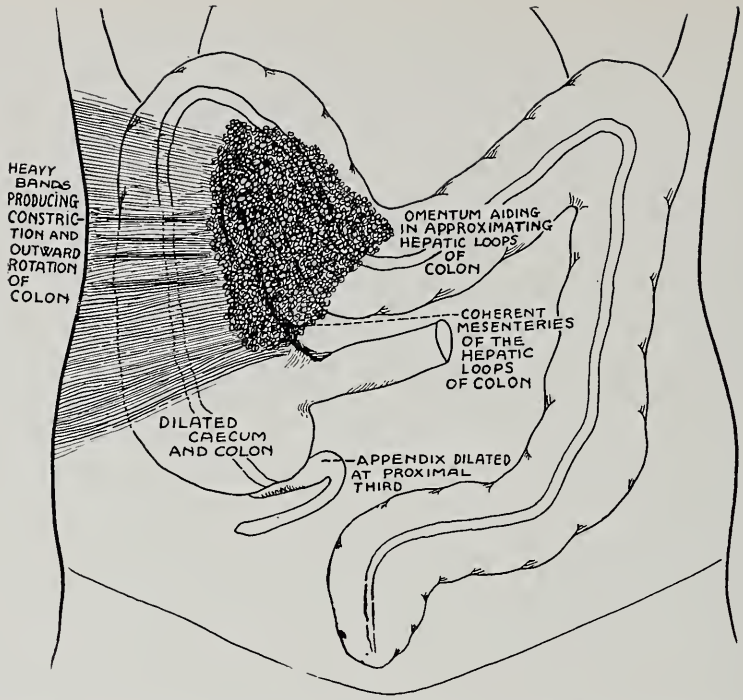
Metschnikoff attributes all the consequences of intestinal toxemia to stagnation in the large intestine. He says: "The contents of the ileum are impeded in the progress through the ileocecal valve on account of the filled cecum. There is even a regurgitation or reflux; and bacteria which the small bowel is unused to, enter. The protecting wall of the infected stream is unable to prevent this systemic damage due to the presence in this part of the alimentary canal of bacteria whose habitat is the colon; and this is the evil consequence of stasis. So long as these bacilli remain in the colon, no great systemic harm results; but the evil is soon felt when the reflux occurs."

Even a dilated duodenum can result from the contents of the bowel damming back into this portion of the alimentary canal; and this part of the bowel may be invaded with microbes from that portion of the colon where the stasis begins. The first part of the duodenum is where ulcer is usually found, and is the one mainly affected. Disease of the biliary and pancreatic ducts may be an associated complication. This infection of the duodenum may result in pyloric spasm with consequent gastric retention and dilatation, owing to the inability of this sac to empty itself before the next meal. A chronic prolapse and dilatation are a sequence, and when the transverse colon is loaded, it may be pulled out of place as well.

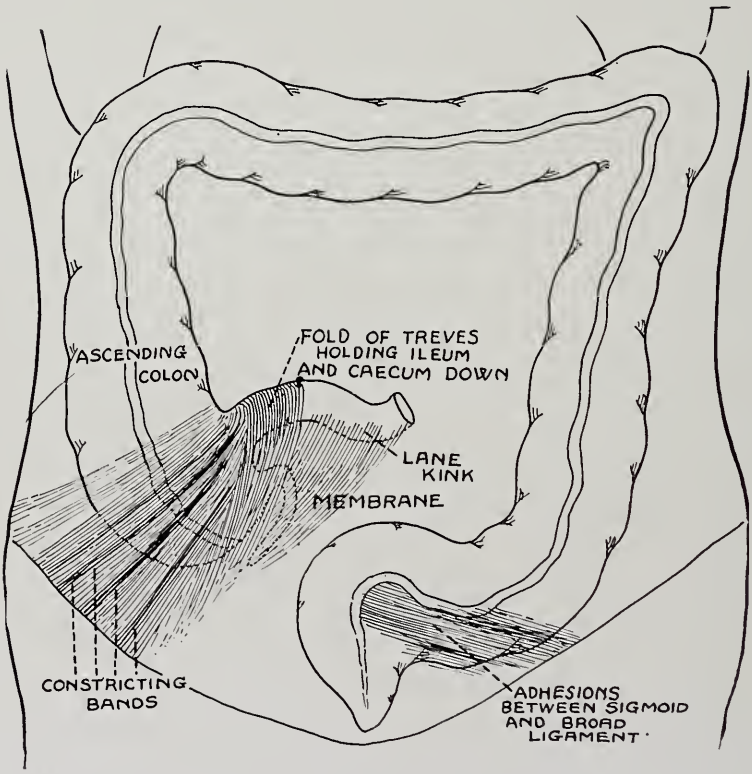
When the intestinal contents are delayed too long in a distended congested loop of intestine, the bac-

Membranous pericolitis with Lane's kink. Pronounced symptoms of intestinal stasis.

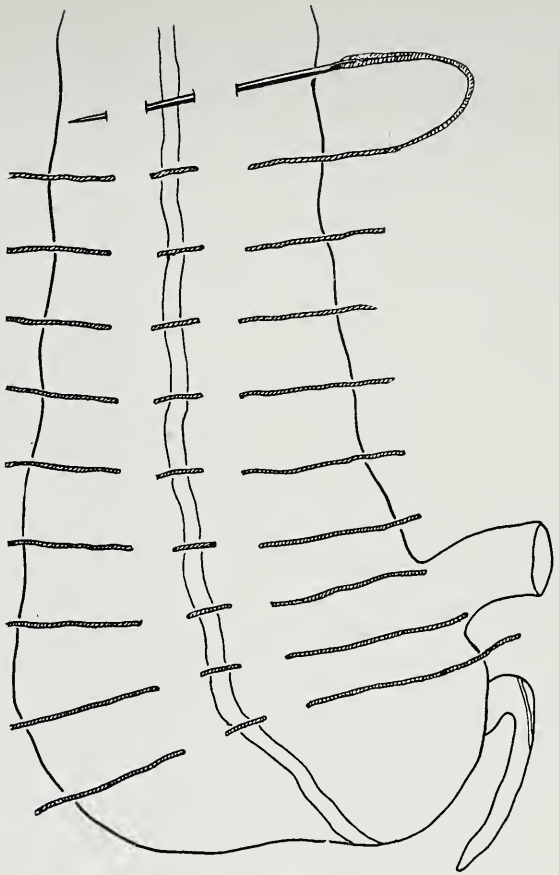
Double-barrel shotgun deformity of colon; appendix included in membrane. (Pitcher.)



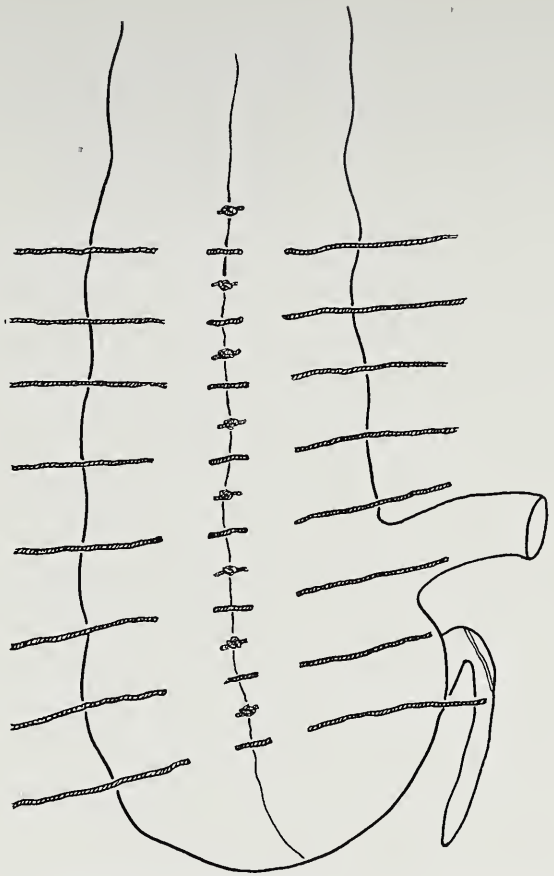
Case of pronounced hepatic kink. Intestinal stasis. Union of transverse and ascending colons. Bowel liberated. Cured.



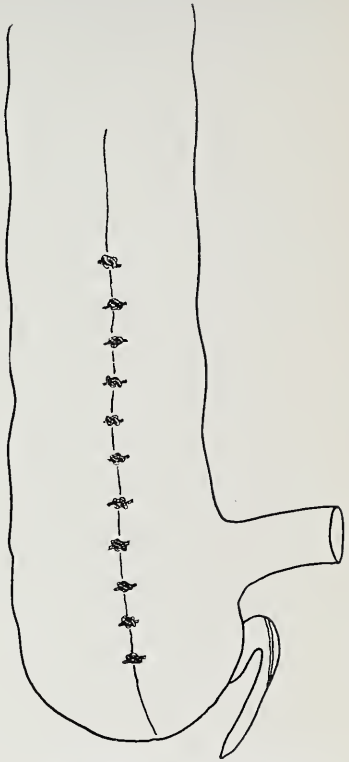
Membranous pericolitis with stasis. Bands severed, raw surfaces covered. Cured.



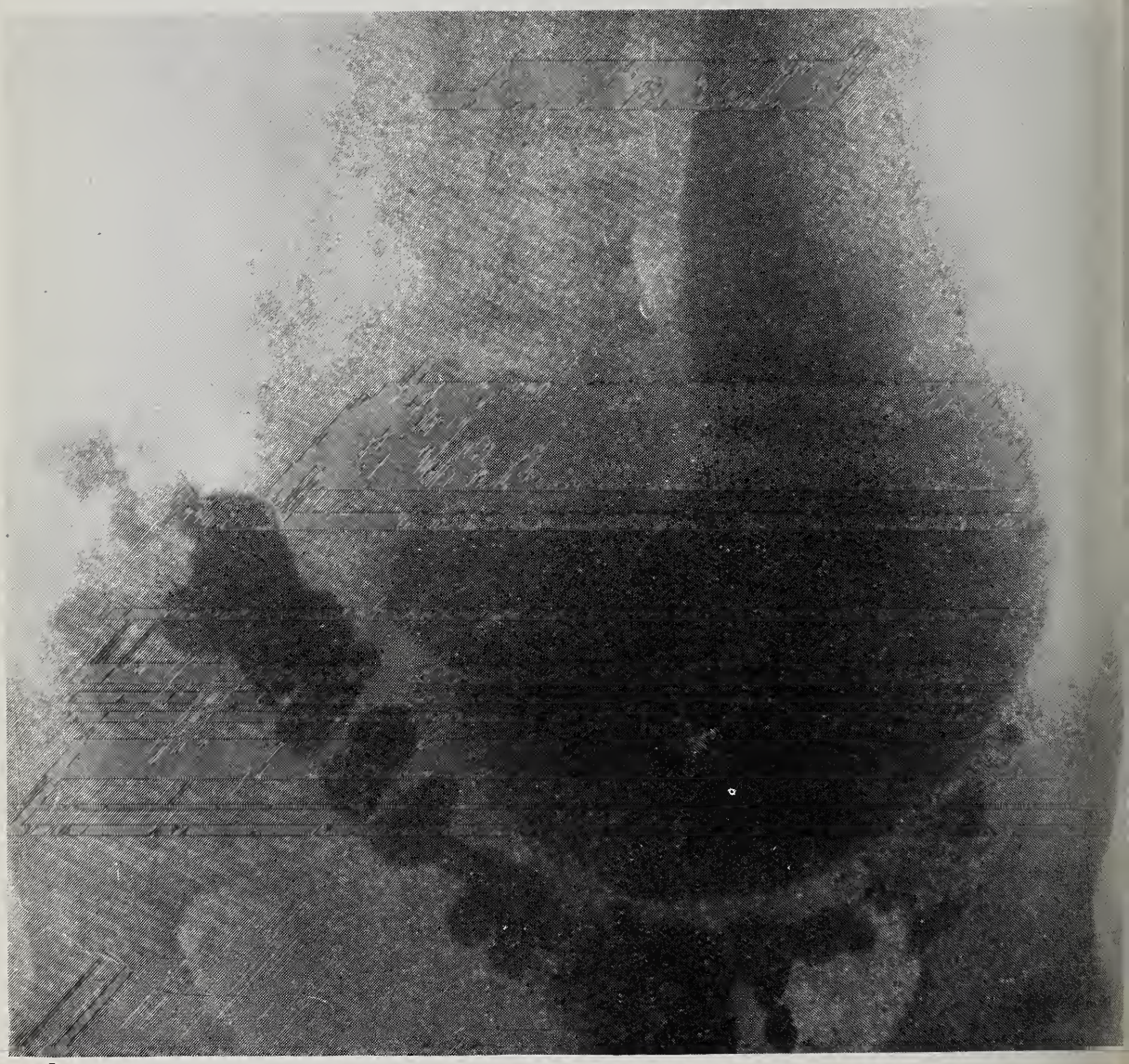
Plication of cecum for dilatation. First row of stitches.



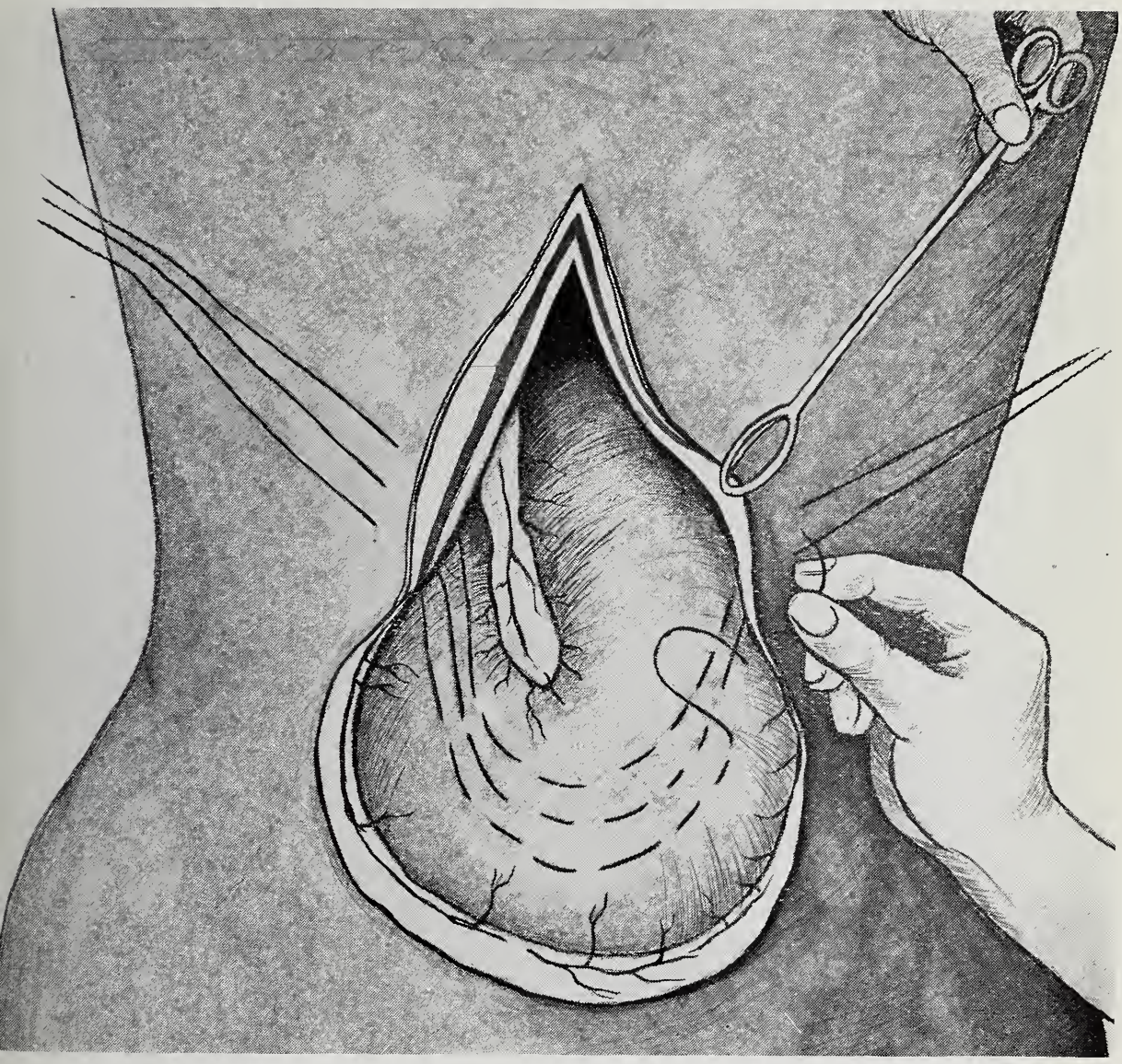
Plication of cecum for dilatation. Second row of stitches.



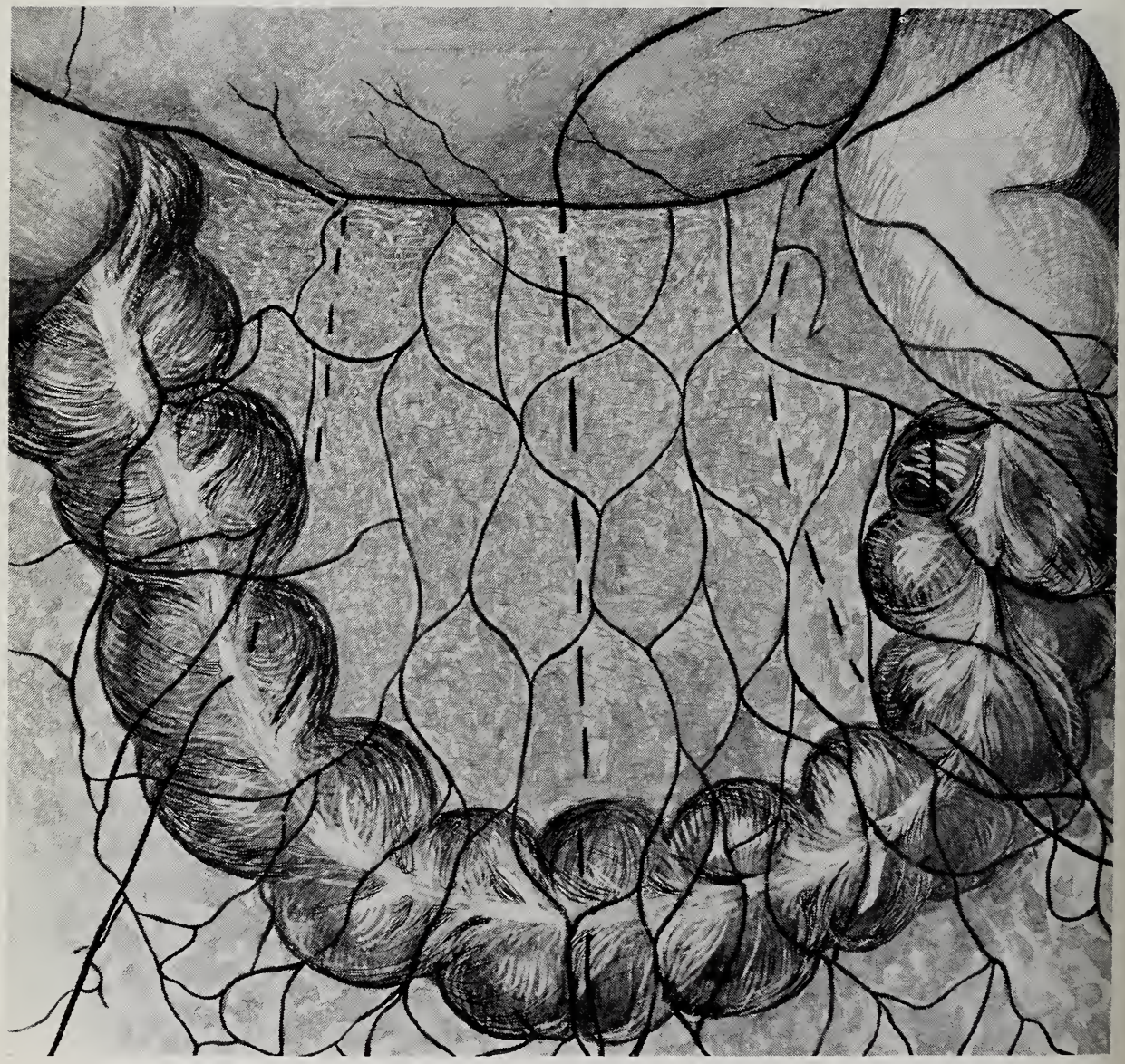
Plication of cecum for dilatation. Last row of stitches.



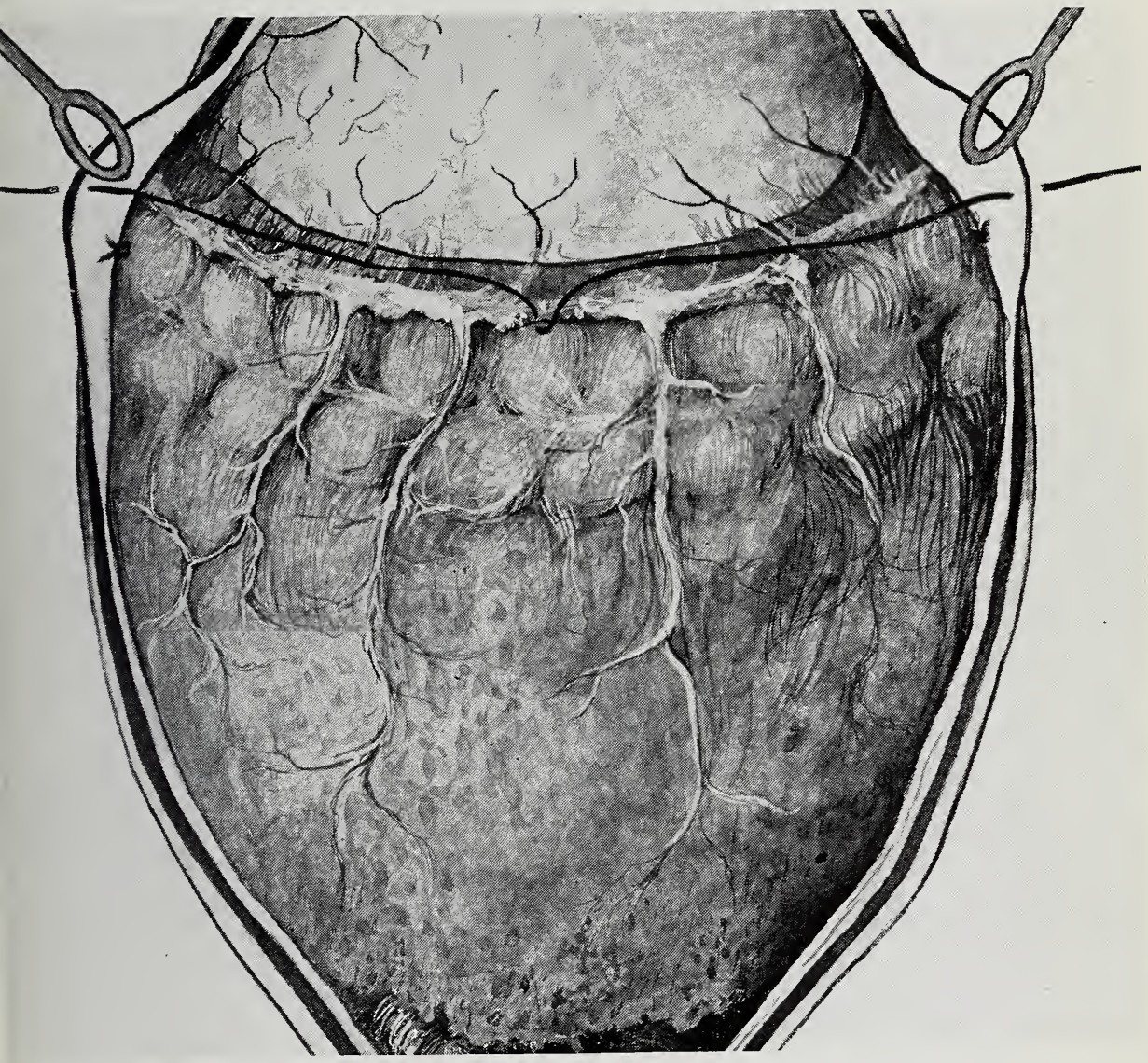
Prolapsed stomach and transverse colon with pronounced symptoms of intestinal stasis. Corrected by Rovsing operation. Satisfactory results.



Elevation of stomach, sutures in position.

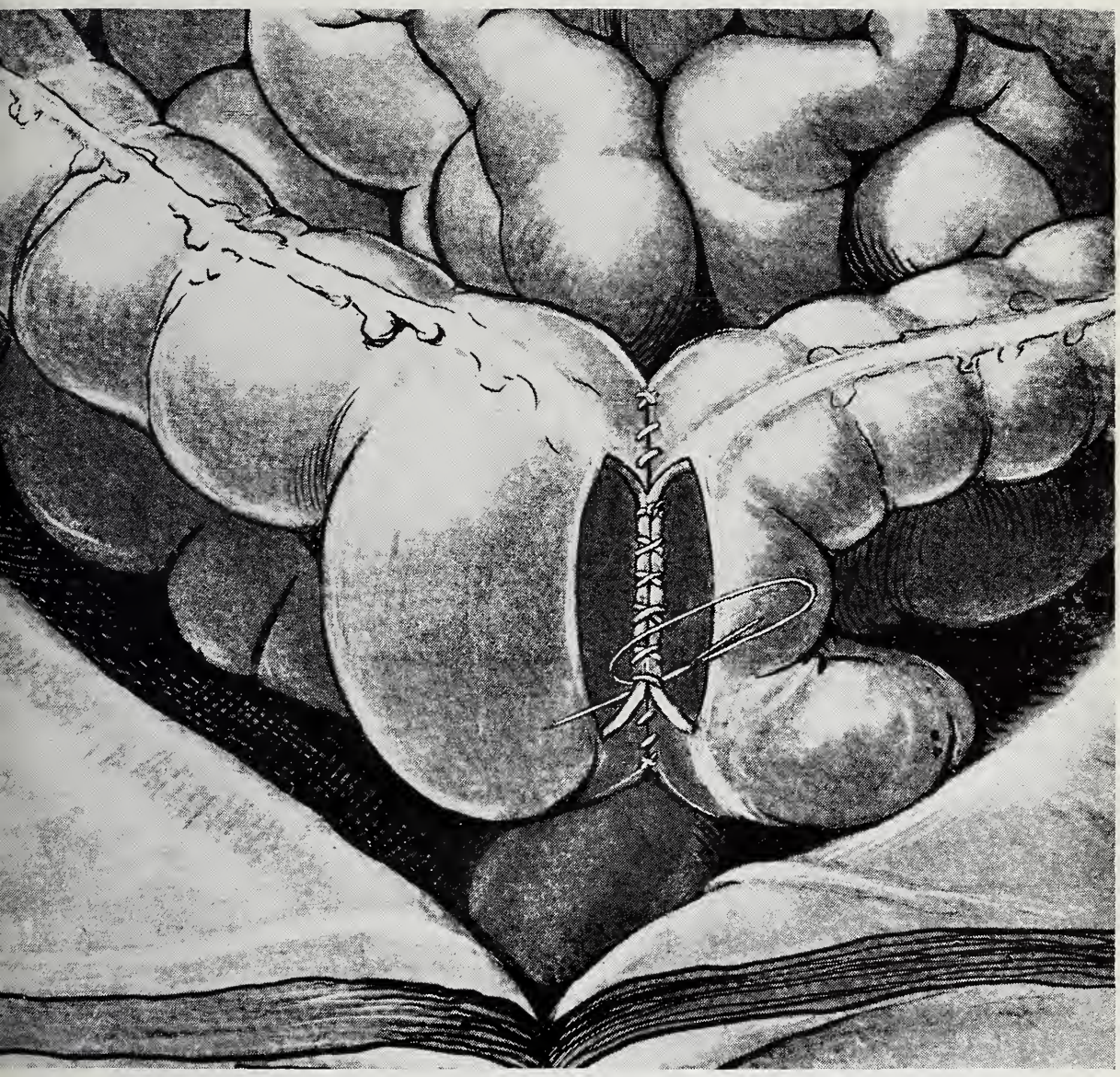


Sutures placed in Rovsing operation.

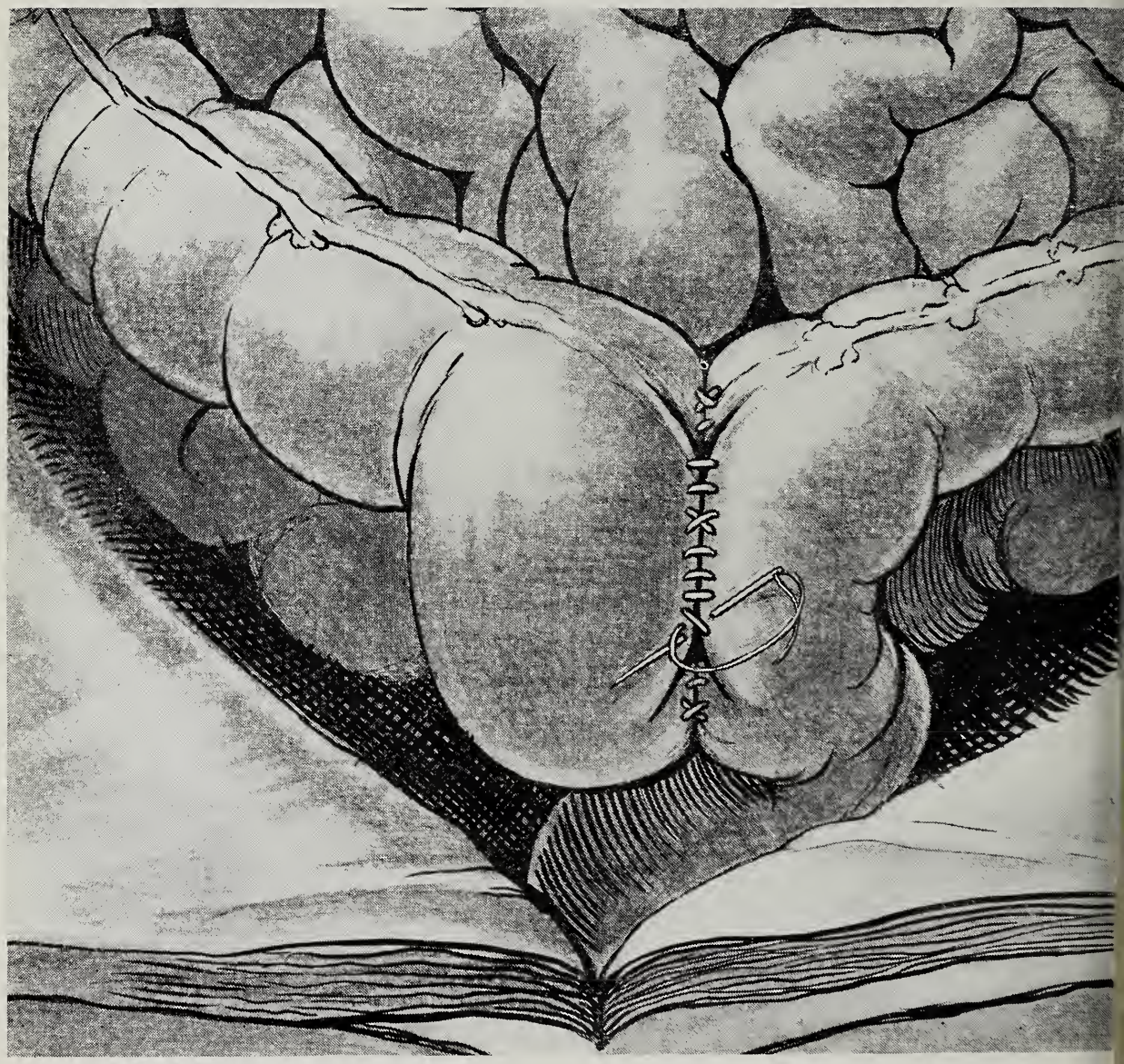


Placing of sutures for elevation of transverse colon.

Case of dilated duodenum and duodenal ulcer caused possibly from intestinal stasis.



Cecum-sigmoidostomy for pronounced colitis and intestinal stasis. First stage.



Cecum-sigmoidostomy for pronounced colitis and intestinal stasis. Second stage.

teria have an opportunity to escape into the lymphatic or the blood-stream, or to migrate through the thinned-out intestinal wall, resulting in a low-grade peritonitis with the production of adventitious bands, which may be nature's method of limiting the destruction by endeavoring to confine the infection to certain areas, but which, in reality, result in attaching contiguous loops of intestine, as the ascending and transverse colon, making a distinct kink so often seen by *x*-ray and at operation.

This membrane and adventitious bands are the natural result of the reaction of the tissue to infection, as noticed in a similar membrane produced in the presence of diseased tubes, ovaries, or any infected organ within the abdomen surrounded by intestinal coils. These bands therefore constrict the colon in sections still more, lessening the caliber of the bowel and furthering the evil consequences. That such conditions do exist, all operators of experience can testify.

An intestine so crippled and limited in its action sooner or later shares the fate of other tissues of the body due to under-nutrition and over-toxicity.

It is therefore further weakened, and is unable to propel the contents onward, greater destruction resulting, as noticed often in the dilated cecum and colon. Loss of muscle-tone or even degeneration of the walls of the bowel itself is a natural consequence. A low-grade inflammation of the mucosa or a pronounced colitis is a natural sequence in certain cases.

Surgeons differ in their opinion about the causes of membranous pericolitis. Most of them believe that these bands are of a congenital origin; but at sometime in the life of the patient affected with intestinal stasis, these bands, which are present along the cecum, terminal end of the ileum, or ascending colon or sigmoid, become significant, because they interfere with

the normal peristalsis of the bowel, act as inhibitors of the normal passage of the fecal contents, and result in stasis and all the multiplicity of symptoms connected therewith.

Sorrell believes that bands, kinks, and membranes cause intestinal stasis.

These bands, which assist in producing obstruction of the large bowel and consequent stasis in the small bowel, are variously named by authors who first especially called the profession's attention to them.

Drs. Daniel Eisendrath and E. W. Schnoor, of Chicago, as well as Dr. Eastman, of Indianapolis, last year reported to this Association the findings of a committee appointed a year previously to determine the significance of these various bands existing in the abdomen and pelvis. The conclusions arrived at by the committee were that the parietocolic fold of Jonnesco is a reduplication of the fold of peritoneum, found during fetal and post-natal life, and that this fold often fuses with the fold of Treves, and that the genito-mesenteric fold of Reid bears the same relationship to Lane's kink that the pericolic membrane bears to possible kinks of the ascending colon. The committee was not ready to report what caused the changes in the pericolic membrane.

Badowe believes that infections of the intestinal contents are "primary factors of greatest importance" in connection with toxemia of the alimentary tract, and the reason for failure of operations for intestinal stasis, is the fact that surgeons are unable to successfully prevent the re-infection of the alimentary canal after operation. This infection begins in early life, and is gradually increased as time goes on. The constant resorption of bacterial products affects the tissues nearest to them, namely, the walls of the bowel. This results in changes in the secretions of the pan-

creas, intestines, liver, and stomach, with a weakened musculature of the bowel and degeneration of the sympathetic fibers. The dilatation may be more manifest in some areas than in others. Resistance of the gut-wall is overcome and bacteria enter the lymphatics or blood-stream.

Daniel, particularly, believes that the formation of bands, constrictions, and membrane is due to local peritonitis, resulting from bacterial activity in and around the intestines. This bacterial invasion of the tissues is evidently from the mucosa. He also believes that, if the origin of the infection is removed, namely, inflammation within the alimentary canal, stasis will disappear. This seems quite rational, but when thick bands, as a result, form and constrict the colon, these bands become of primary importance, and stasis will persist as long as they interfere with the peristalsis of the bowel.

Goldthwaite believes that the poise of the individual has much to do with the position of the stomach and colon. That this is so I firmly believe, for we frequently notice people who stand and sit incorrectly, suffering from this trouble. By standing and sitting correctly the stomach and intestines are not crowded together. Greater space is permitted in the upper abdominal cavity when the lower abdominal muscles are firm and strong, and the upper cavity is made more capacious in long, deep breathing.

Hepatic and splenic flexure, adhesions to gall-bladder, duodenal ulcer, perforating gastric ulcer, or duodenal adhesions or kinks or bands, or obstruction to or along any portion of the colon, sigmoid, rectum, or anus, or sphincter ani, or actual organic obstruction of rectum or an habitual neglect to heed Nature's call for evacuation of the bowel, may eventually bring about stasis.

James T. Case has shown that the ileocecal valve in human beings is so incompetent at times as to allow the return of the colon-contents into the ileum; that, in his opinion as well as in that of Dr. Kellogg, many cases of intestinal stasis are due to the fact that this valve is very weak and that, when this valve acts normally, there is not the reflux into the ileum from the colon, and, therefore, there will not be the absorption from the small bowel of infectious micro-organisms that otherwise would work backward.

The tendency for the bowel-contents to pass onward through the various divisions of the alimentary canal in the normal limit of time, is the rule in a healthy and normal individual; and if there is no hindrance, either from motor insufficiency or from lack of abdominal and intestinal muscle-tone, or from acute angulations, flexures, or constricting bands, to the onward movement of the intestinal contents, no stasis occurs, unless from neglectful habits the colon becomes loaded and the flow of the contents of the ileum is impeded. When stasis does occur, a backward or reverse peristalsis can develop, necessarily resulting in a weakening of all barriers which normally prevent this backward flow. The ileocecal valve, in consequence, also suffers and becomes incompetent.

I do not think that we can attribute to this valve much, if any, congenital incompetency; at least it is almost impossible to conduct any series of experiments or investigations to prove this theory for intestinal stasis. In fact, I believe that an incompetent ileocecal valve is the result of intestinal stasis, and not the cause.

It is true that we may operate upon this portion of the intestinal tract and so re-arrange the valvular barriers, which will for a time at least prevent the reflux of the contents of the ileum from the colon, but how

long will such re-arrangement of the structures act and remain competent to prevent this reflux in case the primary cause or underlying factors are not in some manner corrected or overcome? We must assist the whole mechanism. There must be a removal or correction of any mechanical barriers, such as kinks, bands, or angulations (the latter occur occasionally), due to a general visceroptosis or gastrocoloptosis, and finally assist the patient in building up all the correlated forces which favor regular normal removal of intestinal waste-products.

Treatment.—The diagnosis of chronic intestinal stasis can be suspected from the clinical symptoms; but the definite cause and location of the stasis can be determined only by means of the Röntgen rays or by a rather extensive exploratory operation.

In each case of troublesome dyspepsia or intestinal and gastric disturbances, we should make a series of screen examinations by giving bismuth by mouth, marking the time necessary for its passage onward and through the various segments of the alimentary canal.

A distention of the colon by bismuth enemata will show the size and possible kinks of the colon, cecum, or adhesions. Dr. Payre, of Leipzig, during my visit in 1914 demonstrated an ingenious method devised by him for determining intestinal adhesions by the use of a manganese and iron substance, which he injects into the colon or has the patient take. When located by the *x*-ray in certain portions of the alimentary canal, a powerful magnet placed over the abdomen pulls the intestine up against the abdominal wall, and where there is a pull and pain adhesions are suspected, which interfere with the normal peristalsis of the bowel. Subsequent operations often revealed these adhesions and bands.

When symptoms of intestinal stasis are discovered, proper medical, mechanical, dietetic, and hygienic methods should be at once instituted to overcome the trouble if possible, such as general and special enemas, abdominal massage, encouraging the patient to stand and sit correctly, and to choose clean, plain, nourishing food, used at more frequent intervals and in less quantities, to eradicate all complicating infections, and the use of intestinal antiseptics, if necessary.

But what of the adult individual who having tried all sorts of medical treatment, diet, exercise, position, rest, irrigations, intestinal antiseptics, lubricants, massage, cathartics, etc., is no better, and also who has had demonstrated (by *x*-ray) surgical kinks and great prolapsus of the viscera, especially of the colon and stomach, and who may have had many attacks of intestinal pain, colic, or symptoms of appendicitis, or possibly has passed from the stage of constipation to one of diarrhea with a pronounced colitis, the latter stage being only nature's vain attempt to eliminate the poisonous substances? or what of that case in which nature further attempts to rid the canal of these substances by periodical attacks of vomiting?

One great difficulty is to recognize when it is necessary to perform an operation to relieve stasis, and when to do a late operation, in cases where the end-results have assumed such proportion that the removal of the primary cause would be useless; or, in other words, when degeneration of the tissues has occurred to such an extent that operation would be a failure and the surgeon justified in doing but little, the question arises in these medically unrelieved cases of stasis, what is to be done? It has seemed to me that the radical operation of Lane (colectomy) is seldom justified.

Where there are a dilatation and prolapse of the

stomach and colon or kinks with bands or adhesions, a more simple operation should be determined upon, so that the radical surgical work would be unnecessary.

Rovsing, of Copenhagen, is a strong advocate in these cases of suspending the stomach and colon. He believes many of the cases Lane short-circuits, or does a colectomy upon, are amenable to treatment by his operation. Many other writers have proposed and practiced similar work.

It has always seemed to me a stomach and colon habitually in the pelvis cannot permit the normal activity of the alimentary canal, or give the maximum amount of pleasure and comfort to the owner. I have, therefore, in carefully selected cases with unrelieved symptoms, practised this elevating of the stomach and colon for a number of years with usually a fair degree of success and gratifying results to my patients.

A number of my early cases were done before we had the *x*-ray developed to its present degree of efficiency; and, therefore, I had little to guide me other than the symptoms, clinical history, and physical findings. This suspension was not done according to Rovsing's plan, as it was performed before knowing of Rovsing's method. Of late, I have at times followed his method, except I have not tried to enlarge the upper abdomen in certain cases, as he does, but have, previous to operation, had such patients sleep with the foot of the bed elevated for months, a plan which is usually followed even where an operation is not performed. This accustoms the upper abdomen to the presence of the stomach and intestines, lessens the tendency to stasis, and somewhat enlarges the upper abdominal space, and takes the tension off the lower abdominal muscles. A properly fitting corset worn days also supplements this method.

Other cases of gastrocoloptosis with marked symptoms of hepatic kink, such as "gall-stone colic," with

dilatation of the cecum and more or less pronounced signs of an existing membrane, such as a parietocolic fold of Jonnesco or ileopelvic band of Lane, require surgical attention. In such cases great relief and comfort has been obtained by dissecting off the harmful portion of this membrane, or by severing the constricting bands, and covering raw surfaces by detached omental grafts or with peritoneal tissue, and tacking up the transverse colon so as to prevent the refixing of this part of the bowel to its former location. This is done by suspending the gastrocolic omentum, including the white line of the transverse colon, in three points, two lateral and one central, suspending it from the peritoneum. This elevates the stomach and occasionally obviates the necessity of shortening the gastrohepatic omentum, or for doing a Rovsing operation, in some instances, when the stomach has been prolapsed as well. In cases where the cecum has been very much dilated or twisted, the distal constriction has been relieved, and a plication operation has been done upon the dilated cecum and part of the ascending colon. This very much lessens the size and caliber of the cesspool, and prevents the accumulation of the contents of the bowel at this point. The results have been very satisfactory in these cases.

Adami says of Lane's work: "The nature of the organism responsible for the disturbance and its probable seat of entry should be discovered, and other means of procedure taken before operation is advised. It is unwise to remove the colon; very little is accomplished by this procedure."

Lane undoubtedly gets better results than the average operator. He is a very dexterous surgeon; nevertheless, he has said that the objections are that intestinal adhesions follow, and he reiterates that "if we can solve the problem of intestinal adhesions after colectomy, the results will be marvelous." It has

seemed to me that, in the removal of so large a portion of the alimentary canal together with the omentum, we are depriving the intestine of the necessary fatty protection needed between it and the peritoneum of the anterior wall which limits adhesions and their evil consequences. There can be a recurrence of intestinal stasis from adhesions interfering with the flow of the fecal current. To resect the ileum and unite it to the sigmoid only offers an inducement for fecal contents to back up into the descending, transverse, and ascending colon and cecum. This condition is a positive and realistic objection to such an operation.

Personally I have not thought well enough of these operations to test them out; but for two or three years back I have thought that a short-circuiting operation of less magnitude, and probably one with good results, might be performed by doing a cecum-sigmoidostomy.

Dr. Oppel has demonstrated in a number of instances that, when there is an anastomosis made between any portion of the colon and the sigmoid, there is a damming back of the contents of the colon, and a tendency for the contents to fill up in the loop left out of the fecal circulation, or there is a continuous circle produced through which the fecal contents travel several times.

I have attempted cecum-sigmoidostomy upon animals, as well as on human beings, but have not used it a sufficient number of times to see just what results we are going to obtain in these cases. At the present time I am carrying out a series of experiments to note whether Dr. Oppel's contentions are correct or not. I am using chromic catgut entirely in most of the cases to avoid non-absorbable-suture irritation. I have at the present time in my experience and work, found no evidence of filling up of the loop of the colon after these operations.

CONCLUSIONS

Intestinal stasis has now become one of the most important studies in the field of medicine and surgery.

The symptoms due to this disease are numerous, varied, and complex, so that an erroneous diagnosis is frequently made, and the symptoms present are attributed to other causes.

The disease is occasionally seen in children, and may have a foundation in congenital defects or bands, which tend to inhibit the natural peristaltic action of the bowel.

It is not the presence of infectious micro-organisms within the colon, but the reflux of this infection into parts unaccustomed to the presence of these germs and the ready absorption of the same from these parts, even damming back as far as the pylorus, dilating the duodenum, and interfering with the normal emptying of the stomach at times, that produce the disturbance.

The presence of infectious micro-organisms within the bowel, especially the colon, may result in an inflammation and transmigration of the infectious micro-organisms through the walls of the intestines, producing adventitious bands in addition to the congenital bands present, further interfering with the bowel-action, resulting in obstruction and more pronounced symptoms of stasis.

The incompetent ileocecal valve would seem to be the result of stasis rather than the cause of the same. The diagnosis of this condition can be often made from clinical symptoms present, but a definite location of the kink or primary cause should be determined by the use of bismuth and *x*-ray-screen examinations or skiagrams before operative procedure should be undertaken.

Where there is any tendency or suspected condition which might favor the development of intestinal stasis, the treatment should be begun in early child-

hood, at which time further levelopment of the disease can be often prevented, and the symptoms complained of frequently overcome.

This treatment could be further elaborated in older cases in the way of exercise, massage, medicine, dietetic treatment, and hygienic and mechanical methods, as well as the wearing of suitable clothing, a properly fitting corset during the day, and possibly the elevation of the foot of the bed at night. In pronounced cases not amenable to this treatment, operative procedure should be contemplated.

The radical methods advised by Lane, namely, the ileosigmoidostomy or colectomy, seem seldom justified, as, in the former operation, there results a loaded and filled up, redundant, and useless colon; and in the latter operation, numerous, pronounced adhesions which ultimately can lead to the recurrence of stasis. And, furthermore, these operations, especially the latter, are followed by an unwarranted mortality-rate. The relief of constricting bands and the reduction of unnecessary enlarged pockets, such as dilated cecum, by means of the plication operation, and the replacement of the prolapsed colon and stomach by the mechanical elevation and suturing of the gastrocolic omentum to the lateral peritoneum and to the median incision, and possibly the shortening of the gastrohepatic omentum, and even the elevation of the stomach by means of the Rovsing operation, are simple, free from danger, and prevent the recurrence of these kinks, and favor the normal flow of fecal contents.

This treatment, followed by dietetic, mechanical, and hygienic methods in the majority of cases, brings satisfactory results. In other more pronounced and suitable, well-selected cases, the operation of cecum-sigmoidostomy would seem rational, and possibly satisfactory, but further investigation, research, and experiments should be followed up to determine whether

this will be satisfactory to the patient, as well as to the surgeon.

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DISCUSSION

Dr. J. Frank Corbett (Minneapolis, Minn.): It is with considerable trepidation that I come before you to discuss a subject that is as yet so far from being settled.

In following the results of the short-circuit operations in clinical cases and in experimental operations on animals, I find that great disparity exists,—that is, the results in clinical cases are different from the results in animals. While we might attempt to explain this on the grounds of difference in anatomy between the dog and man, or ascribe it to the fact that the movements of the large bowel in these species differs slightly, these discrepancies do not explain the widely differing results. The cause of this must be attributed to some other fundamental condition. I believe it to be this: In the animal we are operating on a normal bowel, possessing normal motor functions and a normal physiology; in the human these operations have usually been done in individuals where most of the tissues were no longer normal, but were pathologic, and did not possess a complete physiological function.

This may explain why these operations when done with a nearly normal bowel, are not as satisfactory as when they are done in the presence of marked pathology. We are indebted to Cannon, to Rieder, to Holzkneck, to Case, and to other physiologists for our fundamental knowledge of the normal-bowel motor-phenomena so often misinterpreted as pathologic.

In regard to intestinal stasis: I will not attempt to bring anything new before you after the extensive discussion that this subject received at the last meeting. However, I made something of a study in the dead-house of the lesions associated with or causing intestinal stasis. In about 80 per cent of all cases where deformity of the bowel was present, so-called evolutionary bands were associated. These were usually multiple. The condition of the muscularis and mucosa did not suggest that a very great deal of the function had been permanently destroyed. In about 10 per cent of cases inflammatory lesions distorted and obstructed the bowel. These cases were marked by a greater destruction of muscularis and mucosa.

In regard to the Lane colectomy: I think I am safe in saying it is the opinion of most American surgeons that the end results in 50 per cent of the operations have been unsatisfactory. This has been attributed to adhesions from the loss of omentum, and is probably due to that cause. To avoid sacrificing the omentum, the Eastman operation, familiar to you all, has been devised,—a short-circuiting operation.

More recently an operation has been described by Lardinnois. This consists in removal of the omentum from the transverse colon before proceeding to the ligation and excision of the colon, leaving the cecum and the terminal ileum. An anastomosis between the head of the cecum and the beginning of the sigmoid flexure, completes the operation.

Certain disadvantages and certain advantages, on physiological grounds, have been mentioned in regard to this short-circuit route in contradistinction to colectomy.

The men who do not advocate this form of short-circuit of the bowel, say that you may not have a competent or normal ileocecal valve,—and that, because of Lane's kink or for some other reason, the function of the ileocecal valve may be destroyed. They say that the damaged pathologic cecum is a residual cesspool for infection.

The men who favor this short-circuiting answer that objection on the following grounds:

That the drainage of the cecum from the free end of that viscus prevents that from being a cesspool. It relieves the distention of the cecum and gives opportunity for that organ to return to normal. Further than this, they urge that it preserves the ileocecal valve and much of the pain suffered by patients who have been through the Lane operation, has been attributed to the absence of the ileocecal valve.

Further than this we have been taught—and this is empha-

sized when you go back through the history of the cecum—that the cecum has a distinct function, that it concerns starch-digestion, that about 10 per cent of the nourishment of the body is obtained through the function of this area that we sacrifice in the Lane colectomy.

We also know that the terminal ileum has great absorptive powers for other substances. It is rich in lymphatics and is primarily absorbent.

Dr. Carl E. Black (Jacksonville, Ill.): I have enjoyed this paper very much, but I feel utterly incompetent to discuss the subject. I feel that I have not settled down to any surgical procedure in the matter of "intestinal stasis." In fact, "intestinal stasis" has seemed to me a sort of cloak like the old-time use of the word "malaria," to cover up a multitude of things which should be more carefully differentiated.

I have had no experience with the Lane operation as an operation for stasis; nor has it ever presented itself to me as offering such promise as to induce me to undertake it. The only occasions upon which I have removed the colon have been those in which the local pathology made it necessary to do so.

I am convinced that we shall make fewer mistakes if we maintain the local pathology as our guide. Nor do I think we should tack up a loop of colon simply because it is lower than we think it should be. In 1911 I had the privilege of presenting before this association a large number of illustrations taken from cases presenting abnormalities in length and position of the colon. Most of these were from the post-mortem room, although a number of them were from operating-room observations. They showed, however, that many patients had passed through a long and useful life with what would ordinarily be considered a much-displaced or otherwise abnormal colon.

I have tacked up portions of the colon a number of times. I question whether one long loop of transverse colon hanging down in the abdomen is not a better tube through which to pass the intestinal contents than three or four short loops after you have tacked them up.

In the previous paper, above referred to, I reported to you a case which had an unusually long loop of sigmoid. The history showed plainly that there was no stasis in this case until this loop became densely adherent in the right iliac fossa on account of an old appendicitis. An operation on account of the long loop of sigmoid would not have been justifiable, and

the dead-house shows a great number of cases where there was an extraordinary development in either the transverse colon, the sigmoid flexure, or other parts of the intestinal tract without any history of stasis.

Unless there is a pathological condition, such as adhesive bands, which constrict and immobilize some part of the colon, I would not expect to get much benefit from an operation made for tacking up a long loop. Many of these long loops are congenital deformities.

In those cases where there are constrictions that produce stasis it is evident that an operation is indicated. But if we did not have something of that kind I have never been able to see why we expect to improve the patient's condition by tacking up here and there a loop that looks extraordinarily long or not placed just where we think it should be.

We are certainly changing our ideas as we get a larger number of operation-room observations regarding the intestinal canal; in fact, we are only beginning to understand what a normal intestinal canal is.

I have recently had a case of a business man, fifty-five years old, who suffered from intestinal stasis for months. The use of bismuth and the *x*-ray showed that the stasis was in the descending colon. The abdomen was opened, but the only thing found was an area of fine adhesions and chronic peritonitis at the junction of the descending colon and sigmoid. These adhesions were broken up, although they seemed almost inconsequent. Nothing else was done. The patient has been much better, although at the time of operation I felt no confidence in the procedure. Subsequent events, however, seem to show that these adhesions were sufficient to interfere with the proper fecal stream.

I was much interested in Dr. Benjamin's paper, but do not feel that I have really added anything of importance to his excellent presentation.

Dr. J. Rilus Eastman (Indianapolis, Ind.): Stasis, according to the dictionary, means stagnation, nothing more and nothing less.

Mr. Lane gives to the term "stasis" an arbitrary meaning. He says that intestinal stasis is a condition in which we have absorption of toxins from the large or small intestine, due to arrest of the procession of the intestinal contents. In other words, for example, he says that colon-stasis is obstipation with intestinal toxemia; and, at the risk of being jerked up for *les*

majeste, I will say that I think Mr. Lane's term is a poor one, and that it is to no good purpose to give an arbitrary meaning to a simple English word. If we mean intestinal toxemia, why do we not say intestinal toxemia, for the meaning of that term is manifest?

I think we should divide stasis, if we are pleased to use that term, for obstipation, or the old term "constipation," which means just as much as stasis does—I think we ought to divide it into two groups, which may be called *functional* stasis and *organic* stasis. That is, unless there are demonstrable lesions, unless we have constricting membranes or bands, or kinks, unless we have demonstrable pathology, which can be corrected by surgical interference, then we might speak of the condition as functional stasis, and such cases of functional stasis, I believe, should, in the main, be subjected to non-surgical treatment.

Goldthwaite tells us if the individual stands properly, that is, does not stand with the shoulders forward and the chest sunken in so that the lungs are collapsed and the diaphragm displaced downward so that there is encroachment upon the intra-abdominal space, there will be room for the viscera.

If the individual stands from childhood to old age with the abdominal muscles taut, so that the abdominal viscera are held back upon the shelf described by Goldthwaite and F. H. Martin and Robert C. Coffey, the condition of colon stasis for which we are making all of these radical surgical operations will likely not develop. Goldthwaite says that there is no good in suturing up a stomach, for example, after Rovsing unless the individual has been taught to stand so that there is some room for the stomach after we put it in position. In other words, Goldthwaite says there is no use to put the peg in before we have made a hole for the peg. We must prepare the hole first, and then put the peg in.

I asked Mr. Lane if he used alboline after colectomy. He said yes; he used it for thirty or sixty days. I had not the hardihood to ask him whether he used alboline for sixty days before the operation.

Certainly, some of these cases of functional stasis can be relieved by vegetarian diet, by proper exercise, such as horseback riding, and by the prolonged use of alboline. A good many of them can avoid dangerous surgical operations by commonsense non-surgical treatment.

I do not think we can tell much from a skiagram. If we look at the average transverse colon as it is shown upon the

skiagram we will see that the transverse colon hangs like a garland between the hepatic curvature and the splenic angle.

One rarely finds a transverse colon as it is shown in Gray. The simple finding of a descending transverse colon in the shape of a letter V does not mean anything as to real pathology at all.

But by screening and repeated skiagrams we can determine a great deal. In most of these cases of descended transverse colons we will find that there is plenty of play in the movement in the ptosed segment, it will there swing in a caudad and cephalad line and like the pendulum of a clock. If the colon is ptosed and also fixed then pathology begins. There is an important association between chronic colitis and colon stasis.

At the German Surgical Congress for 1910, Payr showed how the stagnation of feces in the transverse colon will lead to colitis with ulceration and Virchow in 1853 showed that these decubitus ulcers produced by the retention of the fecal contents will lead to peri-intestinal inflammation with formation of fixation adhesions.

Fixation of the transverse colon, or any other part of the large intestine, will lead to stasis, because it arrests the normal intestinal movements.

So there is a very important inter-relationship between chronic colitis, intestinal stasis or colon stasis, peri-intestinal inflammations and ptosis.

The first operation of typhlo-sigmoidostomy was made by Von Bergman for volvulus of the ascending colon.

And there is a very important point. Von Bergman made this operation because he had an actual obstruction of the ascending colon. If we make the operation as Dr. Corbett explained, when we have an obstructing lesion we will get good results from it as a rule, but if we make it in cases of purely functional stasis and not exclusively in cases of organic stasis we will surely meet with disappointment.

Dr. Jabez N. Jackson (Kansas City, Mo.,) : It is rather interesting to notice the wonderful amount of interest which has been attached to this subject of the colon within the last few years.

I think in the discussion of the matter of colon-stasis Dr. Eastman called attention to one thing that should be fundamental, and it is that we have first got to distinguish between those cases in which we can distinguish any mechanical interference to account for the stasis, probably due to conditions

within the gut, nerve-supply and muscular, and those which show evidence of some mechanical factor.

I am frank to say that, in those in which you cannot demonstrate something mechanical interfering with the gut, in my judgment, there are only two attainable propositions, one of which I prefer to let alone entirely surgically, and leave in the hands of the medical men. The other is, if you are going to do anything surgically you have got to go the extreme limit of Mr. Lane and take out the whole colon, because it is certainly within the field of mechanical obstructions. These extensive operations on the intestine are getting to be failures because they absolutely ignore our present day knowledge of the physiology of the gut.

I am, of course, most particularly interested in those phases of intestinal obstruction due to the pericolonic membrane that demonstrate mechanical interference with the circuit of the gut, and one which I attempted to describe before this association in 1908, at which time I am frank to say most of the members of the association said that they did not believe any such thing existed. I remember Dr. Eastman told me he thought I was moonshining at that time.

After the elapse of a few years everyone can realize that we are finding these areas of mechanical obstruction in the gut, due to kinks, and those kinks do not amount to anything as a rule unless the kink is preserved by something in the form of membrane or adhesion, or whatever you want to call it, that holds the kink in its position.

In dealing with these forms of mechanical obstruction, I want to say one other word. Many surgeons have been describing what they call the Jackson's veil in the last few years in the literature, as a condition which has absolutely no significance whatever.

In London, in July, I saw Mr. Lane operate upon a case of obstructive disease and when the opening was made you could have taken all the organs in the abdomen, if you had wished, and laid them on the outside. He said, "Here is what they call Jackson's membrane."

I want to say that sort of condition in which there is only a thin membrane has no significance, and I do not want my name in any way associated with it.

Mr. Lane either has not read my article or is lamentably ignorant upon the subject or of what we attempted to describe, which is a definite condition, and any one who can demonstrate a mobile cecum or colon under those circumstances has simply

demonstrated what you can demonstrate in every normal condition; and that is, if you pull the colon onto the outside and wait for a few minutes you will find engorgement, which will give you a congested membrane in a superficial way, one in which there is no fixation of the colon. Now, that sort of thing has nothing to do with pathological conditions at all. When you find that, don't snip it off or do anything else with it, and think you are doing anything good.

If we are going to look at these things we want to look upon those cases in which there is demonstrable mechanical interference with the motility of the gut, and there are some of these cases, but they are not by any manner of means so common as the literature of the present day would indicate.

When we come to deal with these mechanical obstructions in the gut the main thing we have to solve, and it is an important matter, is where do they come from? Now, a great many of our friends have been of the opinion that we are dealing here with a purely normal embryological condition, called Jackson's membrane. If that is true, obviously the only logical thing to do would be to do as you are doing. The gut is all right, outside and inside. It is an embryological defect, and there is no reason or logic for a short circuit or doing anything else if that assumption is correct.

I am frank to say I went to work clinically largely upon that sort of an assumption, by doing what seemed to me to be the most simple thing, and that is simply to liberate the obstructive membrane. I found that a certain percentage of these cases were helped and others were not.

Then I came to recognize there was a second factor that I had not recognized, and that is the presence of the obstruction in the ascending colon or cecum for a length of time which was the occasion of the dilatation of the bowel here.

As a matter of physiology the cecum is practically the bulb that starts the initial circuit of the colonic roundabout, and if we have obstruction above and that meets with constant resistance, it finally dilates and we have the so-called mobile cecum in a large majority of cases in consequence of some condition above.

And yet, if that has gone on for a long time until that distention has reached the period of stasis, you cannot expect to relieve it, when you have dilatation in the first part of the circuit. Under those circumstances it seems logical to try to pick up the weakened muscle and render it efficient.

I do not believe cecal fixation is the logical thing; but I do believe in some cases cecal plication is correct.

Passing rapidly from that, I would say this, that the results of purely mechanical treatment have been in many instances entirely satisfactory, we have had some cases where the results were beautiful.

We have another proportion of cases where the results have not been satisfactory, and those experiences make me believe in many instances that we are not dealing with purely congenital conditions. As a matter of fact, we may have, as Mr. Lane, of London, with whom I talked on this subject while in London, believes that that may be the embryologic basis, but there must be something super-added to it before we get a mechanical obstruction. It is possible that inflammation is something that takes place from the inside of the gut, possibly an old colitis. As a matter of fact in that portion of the intestine the largest amount of absorption takes place.

If that is the case, and we reach therefore the idea that we have gotten something on the inside of the gut, some absorption taking place there that has something to do with this, then we are up to the proposition of dealing with that condition on the inside of the gut.

I want to say, as a general rule, when we come to dealing with intestinal stasis, if we go to the extremity of Mr. Lane who does the more radical operation, which has not given results, although it might be logical in some respects, the less we tamper with normal mechanism in the drainage of the gut the less trouble we are going to have, because we know nowadays that there is just as efficient antiperistalsis as there is peristalsis. I think the operation of Mr. Lane should be practically an abandoned operation. You do not find the ordinary type of colon, but you find a colon loaded with old, hard fecal masses. In other words the antiperistalsis would carry it around in a reverse direction and result in possible distention, the possibility of rupture, peritonitis, and all those consequences. In other words, I do not think this operation has a single sound reason behind it.

Hence we come to this operation here, which is an old operation, which I saw first described in connection with these bands by a man in New York whose name I cannot recall just now. When I first listened to that it sounded logical to me, because it appeared if the antiperistalsis brought the stuff around it would still bring it into the cecum, but we found out there did not seem to be any antiperistalsis as far as the

cecum is concerned unless, as Dr. Eastman said, there may have been obstruction above, then sometimes it will come clear around and come through it, so that it does not affect it unless you have an obstruction. I believe, therefore, if we are going to do any considerable short-circuiting operation, that short-circuiting operation should be adapted to overcoming of the condition at fault, and nothing more.

In the majority of these cases, I am frank to say, in nine out of ten, that we are talking about, we recognize that the whole cause of the trouble is in the ascending colon, though we do find in occasional instances some condition up here in the splanchnic flexure, and that occasionally we find them in the sigmoid. If, on the other hand, we are dealing with a case in which there is colitis and in which we have the proposition of drainage, to do an appendicostomy, and flush that gut for a time afterwards is very much more logical.

In the old days when we were working these things, when they first recommended appendicectomy, we were familiar with the fact that in the literature of the subject there were cases in which, as soon as the fistula closed without the reproduction of other symptoms, the patient objected owing to the fact that he was more comfortable with it open. That is because the mechanical obstructions in these cases are not removed.

If there is colitis, remove the mechanical condition; then open your cecum and flush it; and then when the colitis is gone the fistula can close, and get well of its own accord. That is theoretical so far. In my own experience I have had sufficiently satisfactory results by the simple removal of cause of the plication and I am sticking to that until we know more about the physiology, and until I get something better, and am not advocating short-circuiting operations of any sort.

Dr. Leonard Freeman (Denver, Colo.): Dr. Benjamin has given us a valuable paper. I agree with him that, where it is possible to avoid so radical an operation as Mr. Lane's removal of the colon, it should be avoided.

As regards suspension of the stomach, I wish to offer an operation, probably done often enough before, which I have done many times. It consists in suspending the stomach to the round ligament of the liver, with the idea that this affords an easy place to suspend it, allows some movement of the stomach afterwards, and avoids fastening it to the abdominal wall in

a way that renders it immovable and liable to cause intestinal entanglement.

If we decide upon doing an anastomosis of any portion of the alimentary tract we have to remember that there are many difficulties that we may fall into—that it is not the simple process that it sometimes seems.

We may do an end-to-end anastomosis and end-to-side anastomosis, or a side-to-side anastomosis. It is the side-to-side anastomosis that offers the greatest number of complications, and we have to approach it with care.

We have to remember that we must take into consideration (1) the size of the opening between the parts to be united; and (2) the relative caliber of the parts; and (3) the mesenteric pull upon each part.

Kelling has given us a very excellent article upon this subject. I think his conclusions are not generally known. For instance, if the united parts are of the same size, as are my two fingers as I hold them side by side, it makes no difference whether the opening is large or small, the circulation of the contents of the intestinal tract will go on without trouble.

But, if one portion of the alimentary tract is much larger than the other, for instance, if we are uniting the stomach with the jejunum in a gastro-enterostomy, then it makes a very great difference as to what the size of the opening is.

If the opening is too large there is danger that it will not functionate. Instead of trying to make the opening very large, as we have often been taught in the past, and as I am convinced many surgeons do today, we should endeavor to make the opening small, up to a certain point; the rule being that the opening in a gastro-enterostomy or in any other anastomosis of a large part to a small one, should not be larger than the diameter of the smaller part.

This can be illustrated very well, according to Kelling, by means of a handkerchief (illustrates with handkerchief). If the handkerchief is folded once in its center and held at its ends it will form a tube the opening on the side of which represents a gastro-enterostomy opening larger than the diameter of the tube (or bowel). If we now grasp the edges of the opening near the center and pull laterally so as to widen the opening, a fold or "spur" will at once rise into the opening. This spur may obstruct the opening, especially if, owing to some irregularity of the suture, it does not rise in the center but to one side. On the other hand, if the handkerchief is so held that the opening is smaller than the diameter of the

tube no spur will form. This experiment is easily tried and is convincing. The explanation is that the laws of dynamics determine that the larger viscus expands at the expense of the smaller one, thus causing the farther wall of the smaller one to rise into the opening when the latter exceeds a certain size. A spur of this kind if larger or if to one side may easily obstruct the outward passage of the gastric contents.

This explanation accounts, I am convinced, in some cases, for defective drainage after gastro-enterostomies, and would also account for defective drainage in making a lateral anastomosis of the small intestine to the large intestine.

This is not altogether theoretical nor altogether confined to my handkerchief. A day or two ago I operated upon a case in which there was defective drainage of the stomach following a gastro-enterostomy done elsewhere.

After opening the abdomen and examining the stomach, I found the gastro-enterostomy opening at least three inches long. Upon distending the opening a spur rose up in the center in a very striking manner. I attempted to correct this by making a second gastro-enterostomy opening, and it has not been sufficiently long for me to state whether this has relieved the difficulty or not, but it was nevertheless quite apparent to me what the difficulty was.

Another thing that we have to bear in mind is that in uniting the large bowel, one portion to another, we must take into consideration the mesenteric pull which may take place upon these separate parts relative to each other.

For instance, if we make a lateral anastomosis of the cecum to the sigmoid, and the mesentery upon one side happens to be a little short, it has been shown by Kelling that there often results an axial twist or distortion which in a very inexplicable manner seems to stop the drainage of the intestinal tract. He also insists that in a number of cases where the ileum has been joined to the sigmoid and there was disappointment, owing to the fact that the large intestine refilled with fecal matter, this was not so much due to a reverse peristalsis as to such an axial distortion of the bowel.

We should also bear in mind what Dr. Eastman has called attention to—the assertion of Oppel that in these operations upon the intestinal tract we should always endeavor to avoid any sort of a cul-de-sac, unless we make a fistulous opening at the end of that cul-de-sac. Even though it be a very short pouch, only two or three inches in length, it is quite necessary that it should have a fistula at the end.

Dr. Malcom L. Harris (Chicago, Ill.): When we did not know anything about the physiology of the large intestine we thought it was a simple matter, a matter of mechanics more than anything else; but the more we learn about it the more complicated we find it. I have written several articles on adhesions of all kinds of the large intestine. Most of them were written some time ago, when I thought I knew something about it, but did not know very much. But there was one thing which I did say in connection with the intestines which has proven to be true, and the longer I live the truer I find that statement to be.

The statement was this, that the patient is never sick because the bowels do not move, but the bowels do not move because the patient is sick.

I find that that has a great deal of truth in it. While it was written to apply more particularly to the acute conditions of the intestinal tract, still I find by experience it applies to the more chronic conditions just the same. To illustrate, just recently a patient came in to my office from out of town, and said, "Doctor, I want to get something for constipation." To make the story short, I found that she had come to town two weeks before, and was going home that night. All she thought that was necessary was to come to me to get a prescription.

I said "You have been here two weeks, how many times have your bowels moved during the two weeks?" She replied that the bowels had moved only once in two weeks. That she had friends in the city, and wanted to take in the city, the theatres and so on, that she had been going to dinners, etc., and did not think it was necessary to come in.

There was a woman in perfect health, in a way, who had not had a bowel-movement but once in two weeks. She was not sick, but, as I say, the patient is never sick because the bowels do not move, but the bowels do not move because the patient is sick.

Coming to these operations, most of them were conceived from a purely mechanical point of view. It was supposed if a hole was made in the lower part of the organ the contents would run out by gravity. The intestines do not act by gravity; gravity plays no part in the movements of the intestines. It is not a matter of physics; it is a matter of physiology. Until we find some way of correcting the physiology, our mechanics will all go awry.

Attention has been called to these pouches filling up with

feces. They will do it and do it every time you make a unilateral exclusion of the large intestine. The pouch will fill up and the patient will have trouble as the result.

So every one of these operations built on a mechanical basis will be found to fail, as every single one of them has failed. I do not think there is an exception.

Where there is a mechanical obstruction of the bowel we must relieve it, and that is a mechanical proposition. But, as Dr. Jackson mentioned, the point which I tried to emphasize three or four years ago, is, that in all of these cases there is something else, and this something else is the infection through the intestinal wall. Even, after we have relieved the mechanical phases of these cases, they should be turned back to the physician, who by proper means should endeavor to prevent a recurrence of the conditions.

These are matters, as I say, of physiology and not physics.

Dr. William T. Reynolds (Kansas City, Mo.): I should like to make a suggestion. We hear a good deal about the high cost of living and our methods of living. I do not know just about this, but I would like to hear some observations made in the next year and incorporated in a report in some way on a comparison of the intestinal conditions of white and negro patients; and if any members of this Association have to do with a city hospital where they can make definite observations, I think it would be worth while to make the comparison of the method of living, because, you know, the negro likes corn-bread and things of that kind, and there might be something in diet.

Dr. Benjamin (closing): I appreciate the liberal discussion upon my paper. I want to say that most of these points that have been brought out in the discussion were mentioned in my paper, and I do not think that I really disagree with any of the speakers.

I brought this subject up, not because I knew so much about it, but because I wanted you to tell me something more about it. I have been laboring for a number of years trying to settle this question in my own mind, and I thought you would each lend your mite because it is a subject that is causing a great deal of disturbance among the practitioners. We must settle it sooner or later; we cannot get away from these cases. Neurologists are trying to cure them, but the cases do not get well. Medical men are trying to cure them,

but they do not. Mr. Lane says he cures some of them by his operation.

I will say in reply to Dr. Eastman, who mentioned alboline, that I have talked with Mr. Lane about it. He says he gives alboline to his patients, and has often taken it himself, a tablespoonful three times a day; so you see he is trying to avoid a radical operation himself.

I have tried to devise some means of curing these cases and have had them seek the advice of neurologists, pathologists, and medical men; but still they do not always get well.

You are familiar with all the symptoms that go with this disease. Mr. Lane has vividly described them; they present a picture you see every day in your office.

In reference to cases of mechanical obstruction in comparison with those who have no mechanical obstruction: I will say that I had one case which I operated on last winter, a woman twenty-eight years of age. She had a prolapsed condition of the stomach and colon; they were down in the pelvis. The *x*-ray revealed no particular kink other than just the dropping of these organs. I tried everything imaginable to cure the patient. I treated her for two or three years. I knew the family real well. They all had florid complexions, and appeared healthy and well; but this girl had become very weak, anemic, and the skin was very sallow. She complained of no stomach symptoms, but was asthmatic, and had lost much flesh.

I suggested to her the possibility that weakness, and loss of flesh and color, might come from the stomach and intestines, and that we had better lift the stomach and colon up or suspend it. I tried to fix it in such a way that it would not produce adhesions of a harmful character. We did a Rovsing operation. That woman has now regained her color. She has gained in flesh, and feels well with the exception of the asthma, which is troubling her some yet.

What made the change? I had not benefited her by any other means. It seems to me if you have the stomach and intestines clear down, and you cannot by any known means get them up where they will drain, that something else must be done in a mechanical and operative way.

This case is only an illustration of a number of cases that I have had. I believe, as stated by Dr. Harris and Dr. Eastman, that we should as a rule operate upon these cases, which do not have any apparent mechanical obstruction, only when nothing else will do.

I will say in regard to Oppel's theory that I referred to in my paper, I am carrying out these experiments now to prove whether his theory is correct, but we must take into consideration the mechanical and physiological, and recognize that there is some mechanics to the operation of the bowel, as well as physiology, otherwise the operation of gastro-enterostomy, which is founded on mechanics, not on physiology, would be incorrect.

We must recognize the law of physiology, as well as of mechanics, in this as in many other surgical problems.

TUMORS OF THE MEDIASTINUM

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The classification of tumors is one of the most changeable and unsatisfactory chapters in surgical pathology; each text-book contains a different classification and each author, like the housewife with her sewing-machine, thinks he has the best. There is, however, an encouraging note in the wide discrepancies contained in books published within the quarter of a century just passed, in that, with the increase of our knowledge concerning causal factors in the production of tumors, there has come a gradual diminution in the number of morbid conditions formerly known as tumors. This better comprehension of production of tumors has resulted in the combining under one head of a number of conditions which were formerly considered as independent. Uppermost in this evolution is the recognition by investigators that tumors are made up of tissues normally present in the human body, i. e., the new growth is but a new arrangement of old structures. This does not imply that the new growth is made up of tissues identical with its immediate surroundings, but that the component parts may be found existing normally in the body; chondromata occurring in glandular tissue, dermoid cysts of the ovary, and numerous other examples will come to mind wherein totally unlike "foreign" tissue has been found in tumors, but upon examination we find such foreign tissue exists as such elsewhere in

the body, and we leave to the imagination the task of explaining the presence of such tissue in an unusual location.

By far the greater number of intrathoracic tumors are located in the mediastinum. Save aneurism, they nearly all have their origin in the glandular tissue contained in this space. Neoplasms of the chest occurring outside the mediastinum usually involve these spaces in the course of their development. While it is manifest that the site of the tumor will dominate the clinical manifestations which accompany their development, and determine the line of treatment to be instituted, still more importance attaches to determining the true nature of the growth and the effect it will probably produce upon the surrounding structures. The scheme of diagnosis, therefore, should include careful consideration of the early and more or less obscure symptoms, embracing muscular pains, irregular heart-action, difficulty in breathing or swallowing, spasmodic affections of the laryngeal muscles, and pleuritic irritation and cough, with or without effusion.

The following case illustrates some phases of mediastinal tumors:

The patient, a merchant, aged 57, could not recall having had any serious illness until within the past six months, at which time the present trouble began. He has had to get up once or twice each night for the past four or five years to pass water. Six months ago he weighed two hundred and thirty-five pounds, which was about his average weight; today he weighs one hundred and seventy pounds. Four months ago he began to have a distressing cough, although he could raise nothing from the lungs. He sometimes vomited during the effort, and this action was followed by marked relief. Shortness of breath had caused the patient practically to abandon his business affairs. He had taken much medicine, including iodides, without benefit.

On more careful questioning, the patient said he had had pain between the shoulders for a year or more. This was increased after eating or on lying down, and especially made

worse by rapid walking or lifting. He could not lie on the left side.

Physical examination revealed a mottled, brownish discoloration of the skin with prominent veins; the left chest seemed to be slightly fuller than the right; the supraclavicular glands on the left side were large and movable, but not tender; there was a slight bulging at the suprasternal notch, and light pressure at this point caused an intense coughing seizure, following which the patient was hoarse until after taking a sip of water. There was dullness over the left chest, which extended as high as the sixth interspace with the patient in a sitting posture. This dullness changed with a change in the posture of the patient. Dullness behind the sternum extended a short distance to the right, and was continuous with the heart-dullness on the left; the breath-sounds over this area were absent and they were indistinct over the rest of the left chest below the scapula. The heart-action was rapid and irregular, but no valvular disturbance was detected.

Protracted cough and rapid loss of weight had caused my consultant to regard the case as one of tuberculosis; but the absence of fever and the fact that no râles were present, although the disorder had been going on for six months, made it seem more probable that some more serious disease was causing the pain, which had been persistent from the beginning.

The apical dullness was readily explained by the presence of the enlarged glands, while the absence of fever and local muscular spasm ruled out a high Pott's; and this narrowed the probable limits of diagnosis to two conditions,—aneurism or tumor.

The rapidity with which emaciation had taken place (he had lost sixty pounds in six months) caused me to favor malignant growth in the mediastinum as the most probable explanation of the symptoms and physical findings. Fluid aspirated from the left pleural cavity was clear, and the *x*-ray showed a distinct shadow extending from the suprasternal notch downwards a distance of two and one-half inches, and projecting beyond the margins of the bone.

The growth was removed by subperiosteal resection of the inner end of the left clavicle, and the costal cartilage and attached muscles. It was made up of a number of enlarged lymph-glands rather loosely held together. Little difficulty was encountered after exposing the mediastinum, as the mass was shelled out easily by means of the finger and scissors.

There was very little hemorrhage at the time of operation; and, save a troublesome leakage from a large lymph-duct, probably the left jugular, the patient made a smooth recovery, and lived two years after the operation, dying of some brain trouble, but an autopsy was not obtained. The laboratory reported the growth as a lymphosarcoma.

Case 2.—This specimen was removed from a man, aged 55. He presented a history similar to the foregoing, and was in extremis when admitted to the hospital. We attempted to remove the growth by the method outlined above, but, owing to the intimate attachment of the tumor to the trachea, complete removal was impossible. You will observe that the growth, which springs from the remains of the thymus, has permeated the entire thickness of the tracheal wall, and shows vegetations on the lining surface. The tracheal rings have been destroyed by pressure, permitting the walls to collapse to such a degree as would almost occlude the lumen.

As in the preceding case herein reported, the method of attacking this growth gave a very good exposure, and we succeeded in removing part of the tumor before it became manifest that complete removal would necessitate resecting a segment of the trachea.

The patient died within the next twelve hours; and this specimen, a sarcoma, was removed after death.

For our purpose we may arbitrarily divide these growths into benign and malignant tumors, the former group including aneurism, gumma, and tuberculosis, the latter including sarcoma and carcinoma.

Other morbid growths occur in this region, but the above are the more frequent varieties and all-sufficient for consideration in a twenty-minute paper.

Conclusions founded on observations of the natural history of these several growths will best serve us in their early recognition and differential diagnosis. Some of these growths run a much more rapid course than others, some present marked constitutional symptoms and serious impairment of the general health long before symptoms referable to the chest manifest themselves.

Recognition, therefore, of the wide difference in

the general aspect and progress of intrathoracic growths, aside from the special features which in no small measure characterize each case, becomes paramount in the diagnosis, prognosis, and management of these growths. Malignant growths, for instance, as a rule grow much more rapidly than the benign ones, destroying life in from twelve to eighteen months. A notable exception to this rule is found in lymphosarcomata springing from the posterior mediastinal glands. Such tumors may attain an enormous size, and the patient live a long time, death finally resulting in consequence of metastases.

Growths springing from the connective tissue in the mediastinum,—sarcomata,—may attain considerable size without producing symptoms. This is owing to the laxity of the tissue, and the ease with which enlargement may take place in all directions. To the writer's mind this is a valuable point to remember in attempts at localization of chest-tumors. The site of aneurism is more or less fixed, and you will recall that it is in this type of case that we encounter those enormous deformities of the chest, including bulging, erosion, and fracture of the bony cage. Extensive deformity occurring relatively early in the history of intrathoracic neoplasms, may be induced by implication of a bronchus, which causes collapse of the corresponding lung and compensatory expansion of the opposite side. Derangements of the circulation are constant concomitants of mediastinal tumors. They are caused not alone by external pressure upon the vessel walls, but also by the inherent tendency of sarcomata to permeate the walls of the veins, thereby inducing partial or complete occlusion. The effects are manifold, finding expression in edema, metastases, hemorrhagic effusions into the pleura, pulmonary and cerebral apoplexy, gangrene, and death.

Although functional disorders of the heart with modified rhythm and sounds, without discernible valvular or muscular impairment, are the usual findings, cases are recorded in the literature wherein the heart has been involved in a similar manner to those rare cases of malignant breast in which the disease by extending through the thoracic wall affected the heart. Pain in some degree is usually present, but the chief complaint of the patient suffering from mediastinal tumor will be of his inability to get his breath. The pain, cough, and aphonia are annoying but the dyspnea is persistent and terrifying, filling the patient's mind with ominous forebodings. This, the most prominent of the subjective symptoms, is characterized by a wide discrepancy between the amount of exercise and the respiratory disturbance which follows. The writer has seen a patient, the subject of a mediastinal growth, sitting in perfect comfort, bring on a violent spasmodic coughing seizure and serious respiratory embarrassment by merely walking across the room.

From what has been said, it becomes apparent that no one sign or symptom or hitherto described order of phenomena can be said to be pathognomonic of a certain intrathoracic growth, for the cases vary widely; but, by eliminating the ordinary forms of disease in a patient suffering from serious derangement of the mechanics of the chest, one is warranted in making a presumptive diagnosis of mediastinal tumor.

In the differential diagnosis, aneurism stands out pre-eminently for first consideration. The physical signs of aneurism comprise a loud murmur or splashing sound accompanied by a purring thrill, which is imparted to the hand of the examiner when placed on the chest, and interference with arterial circulation, delay, feebleness or absence of the radial, brachial, or

carotid pulse. Interference with the return flow is common to both aneurism and solid tumors, occurring much earlier in the history of the latter than in the former.

Retardation of the radial pulse on one side may be observed in cases where an aneurism is situated distal to the origin of the great vessels given off by the aorta. Sphygmographic tracings are of signal value in the differential diagnosis, and comparative tracings should greatly aid one in definitely fixing the site of the aneurism.

Symptoms dependent upon pressure manifest themselves later in aneurism than in other growths, but, aside from this, possess no particular differences which would serve as aids in the diagnosis. The physical signs of aneurism, like those of other intrathoracic growths, will vary with the time of observation. If the subject of an aneurism presents himself at a time when there is a considerable degree of elasticity in the sac-wall and, above all, fluid contents fill the sac, the classical expansile tumor, peculiar vibratory thrill, and loud tumultuous sounds render diagnosis easy; quite different, however, are the signs after layer upon layer of clots are deposited upon the inner surface of the sac. Instead of a resilient sac, we now have to deal with a thick, rigid wall which limits the production of sounds, interferes with their transmission to the ear of the examiner, and presents the characteristics of a solid tumor.

Although sarcoma is the prevailing type of malignant tumor found in the mediastinum, carcinomata are found sufficiently often to enable us to summarize their leading clinical features. Primary carcinoma in this region has, in the writer's experience, more frequently begun in the gullet than elsewhere, and the symptoms are those of a slowly but steadily increas-

ing difficulty in swallowing. Solids are first discarded; and in the course of a few months the patient rapidly succumbs if not relieved by surgical measures.

Sarcomata in their early history are painless, and increase in size slowly. Owing to these facts the patient does not present himself until pressure symptoms, cough, hoarseness, or dyspnea, drive him to seek counsel.

Rapidly growing tumors, like gummata, are painful very early in their course, and pressure symptoms, irregular pupil, aphonia, and dilated surface veins, and serious right-heart embarrassment soon follow. Growths in the anterior and superior mediastinum are, in a measure, distinguished by pressure exerted by them upon the superior cava and innominate, while interference with the inferior cava or azygos would suggest the presence of a tumor in the posterior mediastinum. Implication of the venous circulation, ciliomotor roots of the sympathetic, recurrent laryngeal, or pneumogastric, relatively early in the history, speak for solid tumor as contrasted with aneurism, which always shows symptoms on the part of the arterial circulation long before venous stasis becomes manifest.

Tuberculous adenitis, leading to caseation and abscess, affects this region, and the enlarged glands must be differentiated from other growths. This condition, like gumma, is ordinarily not difficult of recognition, as in either instance we are dealing with the local expression of a disease which has almost innumerable general symptoms, sufficient numbers of which are usually present preceding the central chest-lesion to readily distinguish them from other conditions found in the mediastinum.

Finally, we possess valuable therapeutic tests which

will aid in the differential diagnosis of both tuberculous and luetic growths.

Patients dead of sarcoma of the mediastinum are singularly free from metastases. Money, an English pathologist, made post-mortems on a number of bodies in which the disease was wholly confined to the mediastinum; and the writer's experience in the dead-room tends to confirm this view. If this observation proves true in any considerable proportion of the cases of sarcoma originating in the mediastinum, it should encourage surgeons in their efforts to relieve by operation a condition which has hitherto been regarded as well-nigh hopeless.

Numerous methods have been devised for exposing the anterior and superior mediastinal spaces. They include trephining, osteoplastic flap, and longitudinal division of the sternum. These and other operative procedures were devised with a view to dealing with aneurisms.

Bardenheuer separated the muscles subperiosteally from the clavicles and manubrium; and then, by dividing the clavicle and first rib on one side, he was able to remove a growth from the mediastinum. The writer has employed this method in two instances; but, while a fair exposure of the field is obtained, there are certain obstacles which should be regarded before attempting the operation. The chief objection lies in the liability of injury to the pleura. Damage done to the circulation in the course of the operation renders the field more susceptible to infection, and, lastly, the operation is technically difficult.

The operation devised by Milton for exposing the mediastinum is less complicated, and is free from these objections; and it gives a more satisfactory exposure of the field for operation. He divided the sternum longitudinally throughout its entire length,

in order to remove a foreign body from the right bronchus, which he successfully accomplished.

American surgeons, notably Curtis, with a view to avoiding injury to the pleura, have modified the operation by limiting the division to the manubrium, at which point the pleuræ are widely separated.

As modified the procedure consists of an incision, which is carried from the larynx downward in the midline to a point opposite the third interspace. The sternohyoid and sternomastoid muscles are detached subperiosteally, and well retracted while the manubrium is being divided longitudinally. In sawing through the bone the saw should be discontinued on reaching the periosteum covering the posterior surface, and a chisel inserted to pry the severed margins apart. This enables one to divide the periosteum safely under the guidance of the eye.

Mayo's blunt-pointed scissors are well adapted for dividing the periosteum. The margins of the divided bone may be retracted two, or two and one-half, inches permitting the free introduction of instruments and fingers. In doing the operation the chief structures to be avoided are the pleura and internal mammary, or its branches. Division of the latter will cause hemorrhage, which is difficult to control.

The writer's experience with the Milton operation has been limited to the cadaver; the exposure, however, obtained by this method, will permit one to ligate the innominate, carotid, or subclavian, and to deal with operable neoplasms in this region.

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DISCUSSION

Dr. Emil G. Beck (Chicago, Ill.): Dr. Harris impressed upon us last night that the sense of touch is essential to the

success of the surgeon; and this is very true in abdominal tumors. It is even more so in pelvic tumors, but not in the chest, because here we cannot feel any tumors, and here we depend more on another sense, the sense of sight. What do I mean by that? We cannot see the tumors on the outside, except when they bulge out, as aneurysms do when they have destroyed the ribs. As a rule, we have to depend entirely upon the radiograph; and I assert that in tumors of the chest the stereoscopic radiogram is the most reliable means of diagnosis. Thus we depend on the sense of sight.

We recently had in Chicago a meeting at which a gentleman from the East showed us on the screen tumors and other conditions of the chest, diagnosing them by means of radiograms. He said it is the sense of sight (inspection) which helps us most in the diagnosis of these cases. By this he meant the inspection of the radiogram, and not the patient. When called upon to comment upon this, I fully agreed with the doctor that a new mode of inspection, namely, the radiogram, has been added to our older methods; but throwing a single picture on the screen is just like using only one eye instead of two, and this will often confuse us more than it helps.

I know of a method which is far preferable, and will disclose the presence of any tumor in the chest. Instead of using a single radiogram, you should make a set of two, a stereoscopic set, which will give you the distinct outline of the tumor and its location in the chest. The single radiogram is the flat picture. The shadows produced by the different structures in the chest lie all in one plane, and one shadow will overlap the other; and they will melt into one large indistinct shadow, which will not give very much information. It is just the same as if you looked at an object with one eye. If a man looks at an object with one eye, but is accustomed to see with both eyes, he will see the third dimension, the depth; but the man who has congenitally only one eye, sees only a flat picture. If you produce on the screen a shadow of the rabbit made by twisting your fingers, there is produced simply a flat shadow, which will give a picture of the rabbit, but no perspective.

But here I show a streoscopic radiogram of an aneurysm, the picture showing distinctly its location and relation to the other structures in the chest.

As far as operations on the tumors in the chest are concerned, they are very unprofitable, with a few exceptions, and

our diagnosis helps us only in differentiatating those tumors which are curable by other means than operation.

I have operated on two cases. In one of these I had the courage (audacity, some may call it) to operate on an aneurysm in the posterior mediastinum. It seemed ideally suitable for operation. If I had known then what I know now, I could have cured this patient. It was feasible to remove it, but I got cold feet and did not finish my operation, although I could have done it easily, as I saw afterwards when I made the post-mortem. The man died on the fifth day from embolism. I had squeezed some of the blood-clot which was in this aneurysm into the aorta.

If I have time, I will say just a few words about this operation. I was forced to perform it because the man began to develop symptoms of paralysis. It is proved by this picture very distinctly that the tumor encroached upon the spinal column, and began to produce compression on the spinal cord; and I resected the three ribs which were partially destroyed, and exposed the tumor. To my surprise it was hard, like a fibroma, and then I began to doubt my diagnosis, although there had been pulsation right along. So we stuck a needle into it, but there was no blood.

My brothers, who had been interested in the case, were present one of them assisting, and suggested that possibly it was sarcoma. I decided to incise the tumor; and, to be very cautious, I put a purse-string ligature around the top of the tumor first, and then I incised the capsule of that tumor, which was very thick, about three or four millimeters. The contents was a blood-clot, so I knew I had an aneurysm, but I had not exposed it enough to get underneath it. I began to remove the blood-clots, and removed two or three ounces of them, and then I was afraid that the blood might come suddenly when I relieved the plug, which penetrated into the aorta. I told my brother that if the blood came, he should tie the purse-string on my finger, and so when we got very near the lower opening and dislodged the clots, all of a sudden a great big column of blood came up, and he tied the string over my finger, and there we stood without knowing how to finish this.

I had my finger in a very small opening, which was not larger than the finger (I had my finger in the aorta), but the ligature was put as high as I could reach, and we sent for my brother Carl to get advice.

Well, it was two or three minutes that we stood there, not

knowing what to do. He said: "Tie the string and pull the finger up, and then close the wound." I did that, and closed the wound.

I had observed this case for about seven months, but since these threatening conditions arose I think I was justified in trying the operation. For three or four days the man seemed to be very well indeed; then he got chills and fever, and after a few days he died, and I made a post-mortem. I am sorry I did not bring the specimen along. It was very instructive,—a pulmonary embolism.

Glands in the anterior mediastinum which occur in tuberculosis, are operable. However, we do not operate usually on tubercular glands in the mediastinum, as we have other means of attacking them. I have been compelled in one case to operate in such a case. It was a tuberculosis of the sternum, with many sinuses. I removed the greater part of the sternum, and there I saw a large mass of glands. To my surprise these glands were removed very easily. I could remove gland after gland, until I got a very deep cavity and exposed the trachea and some of the large vessels. This patient got perfectly well.

I must relate to you another instance, which is very interesting. About twelve years ago, when I began to practice, I had a case of large aneurysm of the ascending aorta in which Dr. Babcock made the diagnosis, and he made the diagnosis mostly on the dilatation of veins on the outside of the chest-wall and the abdomen. There were dilated vessels also in the pharynx.

Professor Senn advised that I use injections of gelatin, and I made fifteen injections. The man got better, but suddenly died, when I made the fifteenth injection, with symptoms of suffocation. He died instantly, in about a half minute. Professor Senn was present, but before we could even attempt to do anything the patient was dead.

The man was jolly when he came to the hospital to take these treatments, and I had to take the sad news to his wife that he died during the injection.

Therefore, the experience which I have had in intrathoracic surgery of this type has not been very gratifying, except with one case of glands in the mediastinum.

Dr. Haines (closing): I agree with Dr. Beck when he says the *x*-ray is a very valuable aid to diagnosis, but I would like to amend by saying very valuable in the hands of an ex-

pert, one who can read and interpret a plate; otherwise it amounts to very little.

An important point to remember in this connection is, that tumors of the mediastinum sometimes diminish in size, or may wholly disappear, after a few exposures to the ray. I think the one thing that will assist most in the differential diagnosis is a study of the natural history of these growths. We listened but a few moments ago to the report of a case in the details of which mention was made of shortness of breath and undue fatigue having been noted a year or more before enlargement in the more superficial lymphatics was observed. In all probability an *x*-ray taken at this early date would have shown enlarged lymph-nodes in the mediastinum, and would have foretold some of the clinical events which were to follow.

The method employed by me in exposing the mediastinum is not the best method; but, as I have described in the body of the paper Milton's method as modified by American surgeons, I shall not discuss it now. I can think of nothing more embarrassing than meeting with an aneurysm in the course of an operation devised to remove a solid tumor from this region; and still one can but sympathize with the unfortunate operator. Aneurysm occurring in the mediastinum may be fairly easy of recognition during the early months of its existence, but after clot-formation has taken place and one layer after another has been deposited, aneurysm may closely resemble a solid tumor; and still I must insist that a careful study of the history of the growth will put you right in all save a small percentage of cases.

In doing this operation, having once put your hand to the plow, you must not look back; it is difficult work, but there is no reason why we cannot make surgery of this region safe by comparison with the conditions we are attempting to relieve.

The tumor, if permitted to grow, will destroy life, usually in a comparatively short time, hence any procedure which offers hope should be welcome.

You will find but little in the text-books or in current literature to assist in the diagnosis, or to encourage operative intervention for relief of tumors of the mediastinum, but in bringing the subject before this Association I hope to stimulate some of our fellows to activity in this field.

HODGKIN'S DISEASE

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I present this paper on Hodgkin's disease, not because of any especially new or valuable information I may possess, but rather because I am deeply interested in the matter, and selfishly I hope to obtain from the discussion more than I can possibly impart.

I am confident that at the present time we stand upon uncertain ground as far as knowledge of this disease is concerned; and I feel that most of us, at least, are still unable to hold positive views concerning it. Methods of modern research, stimulated to the greatest efficiency by repeated failure, have, however, done much toward opening up a way for a possible final solution. It is true that the valuable work of Bunting and Yates has apparently helped to establish a cause, yet much more needs to be done before all question concerning the etiology of the disease is settled beyond a doubt.

Until recent years there has been a tendency to look upon this condition as a leukemia, but, because of the blood-findings, most writers have classified it under the head of pseudoleukemia. At present I find that several writers suggest that it should be classed as neither a leukemia nor a pseudoleukemia, but as a chronic granulomatous process which at times may assume malignant neoplastic characteristics.

Much of the early work done in connection with diseases associated with enlarged lymph-nodes is now

only a matter of historic interest rather than of real value. It was in 1832 that Hodgkin gave a definite account of the condition, and recognized the fact that it constituted a more or less distinct entity. Since then a great many writers have described the disease. It was to Bonfils, however, in 1856, that we owe the first recognition of the fact that some of these cases represent a distinct leukemia, while others do not. In 1892 when, in a classic paper, Dreschfeld gave such an exhaustive account of these lymphatic diseases, and especially of the blood-findings, the uncertainty of classification was to a degree cleared up; nevertheless, lymphatic leukemia, myelogenous leukemia, pseudo-leukemia, chloroma, Hodgkin's disease, lymphosarcoma, and generalized glandular tuberculosis, have not been given clearly defined places, so that each disease is distinctly separable from the other and recognizable as such.

Hodgkin's disease I believe to be a distinct pathologic process due to a specific infection manifesting itself chiefly in the lymph apparatus of the body through dense cicatricial tissue-formation, running a subacute to a chronic course, and terminating fatally, as a rule, somewhere between two and four years. The disease is more common in males than in females, judging by the histories of such cases. While the ratio is given as three to one in favor of the male, possibly this might not be borne out by more careful search through the literature of well-authenticated cases. The usual period for its occurrence is between the ages of twenty and forty. Cases have been reported in children and others in advanced age. The disease commonly first manifests itself in the neck. Reed, in *Johns Hopkins Hospital Report,* 1902, even goes so far as to say: "We know of no case where the pathologic anatomy was described in sufficient de-

tail to permit of a positive diagnosis in which the disease commenced elsewhere than the neck." Simmons, as long ago as 1903, in the *Journal of Medical Research,* reports two cases of Hodgkin's disease which commenced in the retroperitoneal lymph-nodes. Longcope also reports a Hodgkin's disease originating in the retroperitoneal lymph-nodes. Douglas Symmers reports from the pathologic laboratory of a New York hospital a case of Hodgkin's disease primarily of the spleen, and also a gastrointestinal pseudoleukemia. Since then a number of such occurrences have been reported. It is apparent, however, that the vast majority of these cases first manifest their presence by enlarging lymph-nodes in the cervical region.

I do not find that anyone has given a distinct description of a prodromal period in the course of this disease. Judging from my accurate knowledge of one case, I believe many would be found to suffer from some more or less definable symptoms before the lymph-enlargements take place. In the one case of which I speak there was a time antedating the appearance of the lymph-enlargements by at least three months, when the patient, a strong, vigorous, healthy boy of nineteen, complained of a sense of dizziness while dancing or when exerting himself more or less strenuously. This, too, was associated with a sense of fatigue coming on earlier than was customary with him. On account of this he lost his desire to keep up actively in athletic matters, as he formerly had done. These symptoms occurred during the summer months of 1912; and an examination at the time failed to reveal any evidence of a disturbance which might account for such symptoms. Thinking it was of no serious import, and possibly due to a mild bowel-autointoxication, or possibly a slight metabolic disturbance, a blood-count was not made at the time; and

I have regretted this very much ever since. When he returned to college in the fall he wrote that his power of concentration was not so good as previously. He noticed this particularly in his mathematics. As he was smoking considerably at the time, I recommended that he quit it, which he did; and he thought that he was helped somewhat thereby. In the month of November I received definite information that a glandular enlargement had become evident in the cervical nodes on the right side just above the clavicle. In December he returned to Butte. At this time the total white-blood count was 18,000, with a differential fairly normal. The bunch of enlarged nodes was removed from his neck surgically in the month of February or March following, and portions sent to several of our universities and also to Dr. Fraenkel and Dr. Pick in Germany. The consensus of opinion was the nodes indicated a commencing Hodgkin's disease, though Dr. Pick maintained at the time that sufficient change had not occurred to allow of an undoubted diagnosis, and he held out the hope that possibly the glandular involvement was simply the result of some usual infection of the lymph-nodes. A tuberculin test and also a Wassermann, likewise repeated cultures from the nose and throat, were made. The tuberculin test was negative, as was also the Wassermann, and the cultures gave only such organisms as usually are found in that territory.

An interesting fact was, that early in the progress of this condition, before the operation, and while uncertain of the diagnosis, his tonsils were enucleated, and he was put upon large doses of sodium salicylate, in powder form. For the time the enlargement became apparently diminished in size, in fact, nearly disappearing. Unfortunately, the salicylate produced some stomach disturbance, and became intolerable. On

this account it was stopped. Shortly afterward the nodes began to grow again vigorously. It was again tried by other modes of administration; but the results were never so favorable as at first, though the nodes seemed to soften somewhat. Possibly the tonsil removal had as much to do with this decrease in size as the salicylates, though I have thought that the latter was the chief agent in the early reduction of the nodes.

In succeeding months other nodes of the neck and axilla were removed as they enlarged, and microscopic reports were made in each instance. The establishment of the diagnosis was through this means made certain.

The nodes were, as they usually are, quite hard, except that some few softened, undergoing colliquative necrosis. There was no actual caseation, as in tubercular nodes; and the drainage of this fluid material was followed by a moderate short-lived serous drain, then healing. All of the enlargements, it is true, were moderately soft and discrete early, but soon became hard through the formation of dense fibrotic tissue throughout the lymph-nodes. The older ones at the time of surgical removal were almost as hard as cartilage.

There has been but little splenic enlargement in this case; and only in the last six or eight months has there been a decided gastro-intestinal symptomatology. Following the neck enlargements the chest-nodes were first involved, then later the axillary and abdominal.

As to the blood count: both the red-blood cell and the percentage of hemoglobin remained normal until seven or eight months ago. During the past summer the hemoglobin fell to 90 per cent, and the red-blood count to 5,080,000. At that time the total white

count was 29,750, with polynuclears running 95 per cent of the whole, the transitionals and large and small lymphocytes making up the other 5 per cent. In November the last count was made, and the white-blood cells were 44,200; the red-blood count 4,800,000; the hemoglobin, 80 per cent; the polynuclears had fallen to 88.5 per cent; and the transitionals were 3.5 per cent.

At the present moment the chief distress is noted as arising from the growth in the neck and chest. Hearing is somewhat impaired by pressure of these enlarged nodes upon the external auditory canal, and also by a closure of the Eustachian tubes. The soft palate and uvula are moderately edematous with periods of increased swelling. The sense of smell is somewhat impaired. Vision has not been affected in the least. The intellect is clear, but attempts at sustained reading fatigue greatly. The obstruction produced by the enlarged bronchial nodes audibly hinders easy respiration. There is constant cough, and distress from accumulating secretions in the nasopharynx and larger bronchi. The skiagram shows marked involvement of the chest-lymphatics. Periods of nausea and gastric disturbance have occurred irregularly during the last seven or eight months. The appetite remains very fair, and the bowels are normal. From the very beginning of the trouble the skin has been markedly hypersensitive, necessitating the wearing of materials of the finest texture. Anointing with oil seems to give the greatest relief. At present a bath not followed by this oil application leaves the skin dry and cracking. At times itching over the whole body surface produces considerable distress. Often the skin is abraded or torn in his efforts to obtain relief. These lesions remain unhealed for days.

About a year ago he was given one injection of 5

c.c. of serum from a rabbit, the blood of which had been made markedly lympholytic by repeated injections of a mashed lymph-node removed from his axilla. This injection produced a decided temperature and general reaction for twenty-four hours. Following this he seemed subjectively better. Shortly afterwards Dr. Frank Billings treated him by injections of the Bunting organism vaccine obtained by growth from one of his axillary nodes. He remained with Dr. Billings several weeks, and after returning to Butte the treatment was continued for some while. Dr. Billings sent the vaccine to us for such injections. During the entire time of these injections a filtered *x*-ray was given. On the whole there was a moderate improvement for a while. After this the nodes again began to enlarge in spite of the treatment. In the late spring we were forced to grow our own culture, as the one in Chicago was lost.

I believe there was no doubt as to the character of the organism which we obtained. It corresponded in every particular to that described by Dr. Bunting. The dosage was gradually increased to a thousand million every four to six days. While this was being administered, new nodal enlargements sprang up, and the old processes continued to increase in size spasmodically. It was following this intensive inoculation that the polynuclears increased to 95 per cent of the total white count.

Possibly I should have mentioned that arsenous acid and later sodium cacodylate were administered to their limit early in the progress of the trouble without appreciable effect.

Since August last no treatment has been administered at all because of the apparent uselessness of any known method for the relief of his trouble.

Within the last two weeks I received a letter from

Dr. Billings, suggesting the possibility of some benefit from the use of an antitoxin in conjunction with the vaccine. Dr. Billings says:

This polyvalent vaccine has been prepared by Dr. Rosenow, and sensitized by immersion in the horse serum mentioned in my letter. We have been promised the refined concentrated serum in a few days. As soon as I shall have tried it out in the hospital to know what the dosage should be, that will be sent to you also to use in conjunction with the sensitized vaccine. In the meantime, use the vaccine daily, beginning it with a dose of 20 million, doubling it each time, that is, 40, 80, 160, 320, and so on. The number of bacteria in each c.c. sent to you will be printed on the container, so that the dosage may be easily ascertained. After giving it daily for a week, I would give it every three or four days. I do not think it necessary to run the vaccine higher than one billion, or one billion two hundred million, as the dose every three or four days.

As soon as this preparation reaches me we will once again endeavor to accomplish something for the patient.

This case has, however, created in my mind a decided doubt as to whether or not the Bunting organism is the sole or true factor in the production of this disease. It is difficult to understand why an autogenous vaccination up to the point of tolerating one thousand million of the Bunting organisms at each inoculation, might not have put a stop to the progress of the trouble. In this case there was no noticeable effect, either in prohibiting the growth of the nodes or, on the other hand, of increasing susceptibility as judged by the history of progress of such cases in others not treated by vaccination.

That the Bunting organism was found in this case there can be no doubt, but if immunity to it can be established I am inclined to believe it can be accomplished only by following other methods than those

usually followed in the production and administration of a vaccine.

It is true that the constant presence of the organism in Hodgkin's disease is presumptive evidence of its causal agency; but if this be actually the fact it only indicates that much has still to be done to clarify our present-day conception of immunity.

Another remedy has been suggested as a possible means of cure early in the process while it is yet limited to a small area of nodal involvement. This is the use of radium. The benefit which might be derived from the application I can neither affirm nor deny, yet it does not seem to me that greater good might accrue than through the use of the *x*-ray. It will be noted that in this particular case there was strong evidence of a prodromal period, in which there was manifested a general intoxication before any evidence of lymph-node involvement occurred. If this is a fact it is difficult to understand how removal of the nodes when they do enlarge might cut short the trouble; and likewise it makes it quite as difficult to understand how radium or the *x*-ray applied to the nodes might eradicate the trouble. If the infection first occurs in the nasopharyngeal lymphatics, and this allows of an intoxication of the whole body by absorption of toxins from this territory, the rational procedure would most decidedly be in the direction of eradicating the infection at its site of departure. On the other hand, it is not likely that such a seat of infection could exist without a marked involvement of these lymphatic structures. This was not evident in this case, and I judge is not usual in the cases recorded, though the organism of Bunting has been isolated from fairly innocent-looking tonsils in those affected with Hodgkin's disease on a number of occasions.

During the course of the disease the Bunting organ-

isms have been found in the blood, though this as yet cannot be stated as the result of examination in the prodromal period of the disease.

Everything considered as far as I have been able to obtain information from the work of others, and influenced somewhat by my observations of this case, it would appear that if the seat of infection is originally in the nasopharyngeal territory, the organism does enter the system and become generalized even before the first lymph-node enlargements appear. If we might, even as early as the prodromal period, determine that the disease had started through nasopharyngeal inoculation, it would be quite impossible to eliminate it by any application to or method of sterilization of the original seat of infection. An analogous condition to this, illustrating that which I state, is found in syphilis. Even after the chancre has appeared and before the nodal involvement is evident, its removal will in no wise free the individual from later manifestations of the disease, nor will early removal of the adjacent lymph-nodes stop its progress. In this case that I here relate we have left no stone unturned to discover the possible location at which such an infection might have entered; and, as I stated earlier in the paper, repeated growths were made from nose and tonsils, from about the teeth and gums, and even from the cavities that were discovered in the teeth. The question of the nature of the infection and the site of its inoculation must remain open, to be determined only after much more needed information has been added to that which we are now in possession of.

During the last two years a number of writers have contributed valuable information upon the subject, and I have added hereto some excerpts from these writings, concerning the pathology and treatment of the disease.

H. G. Partridge, writing in January, 1914, on Hodgkin's disease, thus describes its pathology:

At autopsy the enlarged glands, wherever found, are much alike. They appear as enormous masses, fused together, some hard, some soft, in many locations pressing on adjacent structures. I find no mention by any writer of involvement of bone, but in one of my cases there seemed to be a mass springing from the vertebral column. The tumors externally are gray or pearly-white, and on section are of much the same color. In the spleen and liver they are of a grayish-white color. Microscopically, the growths present an increase in the reticular connective tissue of the gland, and within this lymphoid cells, epitheloid cells, giant cells, and, most important of all, eosinophile cells. These latter have been looked upon as of great significance in the diagnosis, especially as between Hodgkin's disease and tuberculosis, as in the latter condition, if present at all, they are found in very much smaller numbers than in the tumors of Hodgkin's disease. It may be stated as representing the present-day view, I believe, that the microscopical picture is almost absolutely characteristic and diagnostic. The secondary growths, most numerous in the liver and spleen, are exactly the same microscopically as the glandular enlargements elsewhere found, and are therefore believed to be hypertrophies of the lymphoid tissues normally present in these organs.

Delafield and Prudden (see the new 1914 edition of their "Pathology"), say that Pappenheim's "granulomatous pseudoleukemia" is the type of lymph-node lesions to which Hodgkin's name is now generally attached, and they add:

True tubercles are not present except as an accidental superimposed lesion, but the nodes undergo peculiar changes which are quite characteristic even in an early stage. The alterations characteristic of Hodgkin's disease may involve the superficial nodes of the neck, axilla, inguinal, or cubital regions, or the lesion may be confined entirely to the bronchial or retroperitoneal nodes. In some cases the spleen is very slightly affected; in others extensive necrosis with fibrous-tissue replacement may occur. The bone-marrow shows a moderate hyperplasia. In advanced cases the liver may show a few of the characteristic nodules.

The above writers also say:

An important characteristic of Hodgkin's disease is, that the lesions have a tendency to remain in nodular form and not to destroy the organ involved completely, if we except the lymph-nodes. There is always a severe progressive secondary anemia, with occasionally a high leucocyte count, though usually the alterations in number and proportions of the white cells from the normal are slight.

In Adami and McCrae's (1914) "Pathology," the following statements occur:

Of recent years, with increasing frequency, there have been observations upon the presence of bacilli, either of irregular shape, like diphtheria bacilli, or beaded and presenting metachromatic granules, obtained from the internal organs in various conditions. In most cases further investigations have indicated that these are not the essential pathogenic agents. In Hodgkin's disease, or at least in an important group of cases included under this name, the proof at last appears to be almost complete, the organisms having been gained in pure culture and vaccines made from them having been found to exert a specific action in bringing about cure or at least betterment. . . . Recently, upon this continent, Bunting has gained from several cases (of Hodgkin's disease) pure cultures of a diphtheroid bacillus. The organism at first shows growth only after several days, and then under conditions of partial anaërobiosis; later it grows more freely and under aërobic conditions. These observations have been confirmed by Rosenow, Rhea, and others; and, what is more, arrest of the disease and progressive reduction in the size of the lymph-nodes have been observed following the use of vaccines made from the diphtheroid bacilli. These observations confirm the view that Hodgkin's disease is not a form of new growth or hyperblastosis, but is an inflammatory overgrowth of the lymphoid tissue.

C. F. Martin, writing in 1914 on "Hodgkin's Disease (Pseudoleukemia)," says that the treatment is, in every particular similar to that of leukemia, and consists mainly in the use of arsenic, though this is of very limited benefit, and *x*-ray treatment. This last-named method is only palliative,—it relieves subjec-

tive and objective symptoms, but never entirely cures the disease.

Martin adds that some of Ehrlich's newer preparations, notably arsazetin, have proved of great benefit, and that in one reported case there were apparently evidences of a complete cure.

Arsazetin may be given either by the mouth or hypodermically. By mouth the dose is 0.05 gm. (¾ grain) four times daily, watching carefully for any of the ordinary signs of arsenical poisoning.

Subcutaneously the injections are given as follows: 0.6 gm. (9 grains) every two days in succession, omitting the injections for five or six days, and then repeating them for two days again.

Martin thinks that arsazetin is safer and better than the other new preparations of arsenic.

James D. Morgan, attending physician to the Garfield Memorial Hospital, Washington, D. C., writes, in a recent article, as follows regarding the treatment of Hodgkin's disease:

> Permanent improvement, either from surgery or medicine, cannot be looked for. The complete removal of the glands or their enucleation is invariably followed by recurrence, either at the former site or in some other region. The giving of arsenic in gradually increasing doses, as recommended by Nelaton and endorsed in the writings of Billroth, is still the practice of today, and is one of the very few remedies showing any actual benefit. The *x*-rays should always be tried, as they reduce the swelling and retard new developments: unfortunately, on cessation of the treatment the growths recur, and treatment has to be again instituted.

Lawson and Thomas recently reported a case of Hodgkin's disease that was treated with the Röntgen rays for weeks at a time with only a temporary reduction of the lymph-nodes, the latter becoming larger than before treatment soon after this method was stopped. The patient was then given benzene (benzol) in doses of 5 minims three times a day at first, then

the dose was increased to 10 minims, and this continued for six weeks. Two weeks after the introduction of this treatment the lymph-nodes everywhere showed a decrease in size, and continued to grow smaller after the benzene was stopped. Some months later, examination showed very small lymph-nodes in the neck and epitrochlears, while the others in groin, axilla, and back, had entirely disappeared.

C. H. Bunting contributed a paper on "Hodgkin's Disease" to the June, 1914, *Bulletin of Johns Hopkins Hospital,* in which he said:

> To summarize our picture and revise our theory, Hodgkin's disease is an infectious disease due to a diphtheroid organism, the *bacterium Hodgkini.* There may often be found a primary lesion at the portal of entry. While in some cases the organisms may remain for a long time localized in the vicinity of the portal of entry, in other cases they early gain entrance into the general circulation, and may be widely distributed. The organism and its toxin show a special affinity for lymphoid tissue, and produce in this the characteristic changes of Hodgkin's disease, changes varying somewhat according to the intensity of the toxin, but resulting ultimately in the sclerosis of the glands. There is at the same time an interglandular inflammatory process, at times very acute, but resulting finally in a dense sclerotic tissue. There are also characteristic blood changes in the disease.
>
> The glandular changes can then be considered only as the result of a toxic action, and contribute to the patient's death merely incidentally, when certain gland groups are extensively enlarged. The cells of the enlarged glands, though atypical, show none of the antagonism to the other body cells characteristic of malignant neoplasm.

J. L. Yates, in an article in the same *Bulletin,* confirms Dr. Bunting's views, and, under the head of treatment of Hodgkin's disease, speaks of the importance of maintaining such patients "upon the highest possible level of general physiological efficiency." He also says that arsenic in the form of salvarsan, administered intramuscularly and intravenously, is without

appreciable effect upon the ultimate outcome. Tonics are often of great benefit, "but arsenic no more than iron."

Yates advises surgical removal of the greatest portion of the disease that can be done with safety, gentleness, and thoroughness, in cases where tonsillectomy has first been performed and recovered from. The excision should be as complete as that advocated by Crile in cancer of the cervical glands, and the field of operation should be thoroughly treated with tincture of iodine, and drained. This is the "primary procedure," and under "supplementary procedures" Yates advises the hygienic and drug treatment quoted above, and says, further, that the whole area of operation should be treated by the *x*-rays, in measured doses, begun the same day as the excision, or the first or second day after it, and repeated as often and for as long a time as safety permits.

The same writer adds that, in his experience, vaccine treatment is, as yet, of insufficiently demonstrated value as either a curative or a prophylactic agent, and he says in conclusion:

> A sovereign remedy for all cases is not now conceivable. At present the greatest need is some therapeutic agency to control glands not directly accessible; for once extension, which may occur early, has reached either the thorax or abdomen the prognosis becomes relatively, if not absolutely, hopeless.

Billings and Rosenow, in a recent paper, report favorable results in the treatment of Hodgkin's disease with Röntgen rays and an autogenous vaccine prepared from the diphtheroid bacilli of Bunting and Yates, the vaccine being given subcutaneously in doses of from 6 to 10 millions at first, and gradually increasing the dose to a maximum of 100 millions.

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DISCUSSION

Dr. Emil G. Beck (Chicago, Ill.): I am indeed gratified to have the opportunity to discuss this paper, because I have a suggestion in the treatment which I believe may help the doctor in bringing a cure to his beloved son.

I have recently shown in Chicago before the German Medi-

cal Society a very extensive case of Hodgkin's disease, in which I have used a new method of treatment, and the Society has invited for the discussion and for the examination of this patient the men that have been mostly interested in this disease, including Dr. Billings; and I am going to show you the photographs as well as the radiographs, and give you the theory on which I base this new treatment.

Before doing that, however, I will discuss a few points in the doctor's paper, first, that the disease can be diagnosed with much greater certainty than one is apt to think from the literature or from the confusion that exists even at the present time. The disease is usually mistaken for either sarcoma or glandular tuberculosis; and we know that in tuberculosis the glands suppurate very often, and in Hodgkin's disease they never suppurate. Besides, in the early stage which we call the prodromal stage, the glands sometimes remain small and localized at one point for a long time, and then suddenly begin to enlarge so rapidly that from that alone one can assume that he has to deal with a case of Hodgkin's disease.

The excision of the glands will give us a great deal of information. Microscopic examination, and even a cross-section examined with the bare eye, will serve to differentiate.

I am sorry I did not bring those specimens along to show that the cross-section of such a gland gives you an entirely different picture from that of a tubercular gland. But the microscopic examination clears up the doubt. Besides, the cultivation of the diphtheroid bacillus will confirm the diagnosis.

Another point, we must know that the reason that Hodgkin's disease is confounded with tuberculosis is, that in twenty-five per cent of the cases of Hodgkin's disease, a glandular tuberculosis co-exists.

I have had such a case in which Dr. Hektone and others made the diagnosis of tuberculosis by transplanting a part of the gland into a guinea-pig. The guinea-pig developed tuberculosis. On the strength of this we began to operate, and the more we operated the quicker the glands returned. This is a characteristic of Hodgkin's disease. If you should remove the small gland, mistaking it for tubercular gland, and the recurrence is very, very rapid, and extensive lookout for Hodgkin's disease, and make the examination by means of the cultures.

In reference to treatment: For years arsenic was the most

lauded remedy of this disease, and, I think, it is even now employed.

Since the discovery of the *x*-ray, this agent has been tested in many clinics in Europe. I have read over the reports, which show that, while the results were very gratifying during the treatment, as soon as the treatment was stopped the glands recurred. And not only did they recur, but they recurred with greater rapidity than before, so that at the present time there is not a single case on record in which the *x*-ray has cured the patient; and, so far as I know, there is not a single case on record which has been cured by any treatment. I am very sorry to say that, but I believe in the case of the doctor's son there is a great deal of hope because he stated in his paper there is a necrosis, and it was in my case where there was a liquefying necrosis, and through that I have been able to obtain the means for improving my patient.

My case is that of a young lady, twenty-nine years of age, who started four years ago, with "apparently" tubercular glands, and was operated on at three different times in short succession, and every time when she was operated on the tumors recurred with such rapidity that two months after the operation the tumor grew to twice its former size. I have here two pictures, and you can see by these two pictures the growth as it presented itself to me when I saw it first, in February of this year, 1914.

I did not hope for any cure in this case, because it had progressed so far that the young lady could hardly breathe, due to the obstruction of the trachea; besides, she had obstruction of the esophagus, and the case seemed hopeless. I show here a radiogram taken at that time, and you can plainly see that it involved the upper half of the left lung, and pressed down the heart and vessels, causing other than the respiratory symptoms.

I noticed that there was a slight fluctuation at the top of one of these protruding tumors, and I withdrew a clear fluid with a hypodermic syringe and obtained 20 c.c. I diluted it ten times with salt solution, to experiment on the theory of Prof. Fischera of Rome, who used a similar method of autolytic serum for cancers. I began to inject 2 c.c. every three days; and to my surprise I found that inside of two months the tumor was reduced about one-half. The patient gained twelve pounds in two months, and all the obstructive symptoms disappeared.

I have taken stereoscopic pictures of the patient before

treatment was begun, which I here exhibit. Please compare them with the picture taken recently, and you will note that the external appearance shows at least two-thirds reduction of the tumors in the neck. Likewise the two sets of stereoscopic radiograms of the chest show distinctly the reduction of at least two-thirds of the tumor in the chest-cavity.

She is still under treatment, and the tumor is constantly getting smaller, and she is getting better.

The blood-findings, of which I have kept record, are taken after each injection, and you note that the blood has returned to almost normal. Outside of the leukocyte count of twenty-four thousand in this case, I think there was no marked change from the normal. The eosinophiles were one and a half or two and a half per cent, which is characteristic of the disease.

The diagnosis was confirmed by Dr. Rosenow. I gave him a piece of the tumor for the cultivation of the microörganism, and he has found it and made a vaccine and kept it for eventual use.

During my absence in Europe our autolytic serum ran out, so we sent to Dr. Rosenow for the vaccine. The entire quantity was used, but without any appreciable reduction of the tumors. As soon as the autolytic serum was stopped the patient began to lose, and lost seven pounds in the four months of my absence, although the tumors did not grow any more.

The characteristic thing, however, was that the point where I removed small tumors for the examination, there has been no recurrence, while previously recurrence was immediate after the operation.

When I found we had no more of the original serum, and I could not detect any fluctuation in the remaining tumor, I removed two small tumors from the right axilla. By the way, when I left there was only one tumor in this axilla. During the course of treatment it had separated into two small tumors, connected only by adhesions.

From these two tumors I made an autolytic serum under the most aseptic precautions. I sliced them, as you would a cucumber, and placed them in normal salt solution. After sixteen days in an incubator I withdrew this fluid, and sealed it in small bottles; and this serum is being injected every two days in 2c.c. doses. The effect seems to be the same as with the first serum, which I obtained from one of the softened areas.

I wish I could produce another necrosis, which would give

me the same fluid; but it seems that the autolytic serum has the same effect. Since using the last-made serum she has gained seven pounds, is back to her one hundred and twenty-nine pounds weight, and the blood-changes are normal at the present time.

One point I wanted to make in reference to the doctor's case is, that he has a case with a softened area, and I would advise him to withdraw that fluid, dilute it ten times with a salt solution, inject 2 c.c. every three days; and I believe the boy will get well.

Dr. A. F. Evans (Wichita, Kan.): I wish to report a case that came to my office about six months ago. The patient was a retired farmer, aged sixty-five. When he made his appearance in my office the glandular enlargement of the neck was very marked. The tumors hung down over the low collar which he was wearing; and there were also large tumors in the axilla, and tumors in the groin.

He was under *x*-ray treatment, and was given Fowler's solution. I might say also that he complained of extreme weakness at this time. While under this treatment of *x*-ray and Fowler's solution there seemed to be no improvement, either in his general condition, in his strength, or in the size of the tumors. The tumors remained about the same.

A tumor was then excised, and sent to Dr. Rosenow and Dr. Billings, and a vaccine was produced, and his local physician began giving him those treatments, with a vaccine. However, the *x*-ray exposures were continued.

The tumors apparently decreased somewhat in size under the vaccine treatment, but the patient failed to gain in strength. He was under the vaccine treatment about six weeks, when he suddenly developed an inflammatory condition involving the abdomen. This condition was diagnosed as appendicitis by his local doctor, but on account of his general condition operation was discouraged.

The inflammatory condition in the abdomen continued until the appendix ruptured and an abscess formed. I was called, and concurred with the diagnosis of appendiceal abscess, and recommended operation.

At this time the patient was very weak, I do not know what his blood-pressure was, but the slightest pressure on the radial artery would stop the pulsation, and he was anything but a cheerful subject for surgery.

I made an incision through the right rectus muscle. I

thought of the possibilities that the mass that I felt there might be a broken-down lymph-gland, but with the history we thought it was appendicitis. He also gave a history of having had an attack of disturbance in the abdomen eighteen years before, which I am inclined to believe was an attack of appendicitis.

We made the rectus incision, opened up the abscess, and found quite a large enterolith. This was removed. The appendix was then found, and removed without any great deal of difficulty; and a drainage-tube was used, and the patient was put to bed.

During his convalescence he had rather a stormy time for a few days, but he apparently began to gain considerably. His vitality increased, and the glands began to decrease in size; those especially that were quite large in the neck decreased one-third the size they were before the operation.

It was about a month before the wound through which the appendix was removed healed, and he then was able to get up and move around the house and make short excursions downtown. I was talking to his doctor a few days before I left my home, and he was inclined to believe that the glandular enlargement was becoming more pronounced again.

I would say that during this time that he was confined to his bed from the appendicitis there was no vaccine treatment used. The doctor that was giving the vaccine treatment lived a few miles away from the patient, and it was impossible for him to attend to that part of it.

Whether Hodgkin's disease had anything to do with appendicitis, or whether the injections had anything to do with it, is a matter that may be worked out. I am inclined to believe, however, that it was purely coincident, and that the apparent improvement in the Hodgkin's disease had very little to do with the appendicitis or with the surgery, and may have been a continuation of the results that they were getting from the former vaccine treatment.

Dr. D. P. Mayhew (Colorado Springs, Colo.): I would like to mention a case because of the interest which it brings to us in the early diagnosis of a case of Hodgkin's disease, and possibly shows the value of vaccine in these cases.

Early in this year a young man was referred to me by Dr. Webb, of Colorado Springs. The patient had on his neck, at the side of his thyroid cartilage, on the left, a growth about the size of a robin's egg. It was hard and apparently fixed to

the thyroid cartilage, and the impression given was that of a chondroma.

The man was supposed to be tubercular. I mention this in passing. He gave a history of having had large masses on both sides of his neck like those seen in Hodgkin's disease. These masses had disappeared, leaving behind nothing but the above-mentioned growth.

Under local anesthesia the mass was removed with great care as to the asepsis. It was taken by Dr. Webb, and a culture was made from it, and the typical organism of Hodgkin's disease was found. Guinea-pig inoculation showed no tuberculosis. A vaccine was prepared, which was given to the patient.

Contrary to the usual observation, the growth did not recur, and up to the present date there has been no further sign of the trouble.

The blood-picture, I might say, in this case, was typical of Hodgkin's disease.

The interesting points here are the fact that an apparently typical Hodgkin's disease had spontaneously retrograded, leaving but the one neoplasm mentioned, which lay in a very unusual situation, immediately alongside the thyroid cartilage, to which it was intimately adherent; and the fact that from this neoplasm the typical organisms were recovered.

I think there can be no question as to the diagnosis in this case. Dr. Webb is an exceedingly skillful man both in pathology and bacteriology, and it is on his work that I rely as to diagnosis.

That there has been no recurrence argues the value of the vaccine in at least some cases.

Dr. Witherspoon (closing): I dislike to take more time in the discussion of Hodgkin's disease, but there has been a point raised which is worthy of further comment. This is the one brought to us by Dr. Beck, and concerns the treatment of Hodgkin's disease by the use of the broken-down gland itself.

It will be noted that the doctor uses either that material obtained from a liquifying node in the growth or that which is prepared by immersing the sliced node in normal saline solution under most sterile precautions and shut off from the air. The problems which present themselves to my mind are these: Does the doctor get from this material an aggressin, the result of the growth of the organism under anaërobic con-

ditions, which is normal to it? or does he get some ferment capable of destroying the lymph-gland, which it gradually does do, under the conditions instituted by him? or does he use some portion of the protein structure of the lymph-nodal tissue as an antigen.

It will be noted that Dr. Billings in generating his vaccine, grows the organism under aërobic conditions, and also upon an artificial nutrient medium. As suggested by Dr. Beck, following the idea of Fischera, the organism is grown upon the lymph-nodal tissue, under anaërobic conditions, which would be ideal. It is quite possible that the organism so grown may be used more perfectly as a vaccine than the one grown on artificial medium under aërobic conditions. Secondly, it occurs to me that the doctor may obtain in the solution the presence of aggressins which he uses as antigens by injecting. Thirdly, it is possible that he may have some lytic structure also, which upon injection tends to destroy the lymphatic tissue, thus exposing the organisms present in it more abundantly to the oxygen of the blood, thereby tending to alter them and make them less resistant to destruction in the infected individual.

As the organism of Hodgkin's disease seems to be anaërobic, those remedies which will increase the oxygen-conveyance in the fluids and the upbuilding of the body-tissue are most useful; therefore, iron and arsenic are always given advantageously.

I shall make use of the method suggested by Dr. Beck in the case of my own patient, and shall be better able to talk upon the subject later on.

SPLENECTOMY

Review of Literature; Report of Personal and Other Cases

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SALT LAKE, UTAH

Indications.—The indications for splenectomy are as follows: (1) injury or prolapse, (2) certain cases of movable spleen, (3) sarcoma or lymphosarcoma in the early stages, (4) simple hypertrophy, with or without cirrhosis, (5) cysts, (6) hydatid diseases,—localized tuberculosis of the spleen, (7) splenic anemia with splenomegaly.

Dr. Allen B. Kanavel, in June, 1910, reported a series of experiments on splenectomized dogs, demonstrating "that while repair of broken bone is apparently delayed slightly, for practical purposes we cannot attribute ultimate nonunion to the loss of the spleen." His conclusions were as follows: "The removal of the spleen when it is ruptured interferes slightly with the repair of any fracture, but not sufficiently to contraindicate its removal if the exigencies of the case demand it. On the other hand, if it can be sutured with safety to the patient it should be done. . . . Splenectomy results in impairing the regenerative power of bone in varying degrees in various elements, having in general a more marked effect upon the medulla, but in individual cases this may be but slightly affected, while the other elements may be more seriously affected. In other words, the effect is prob-

ably the result of the general change in the system rather than any specific effect upon the bone."

Contraindications.—As a person can live in comparatively good health without a spleen, provided that his other organs are in a healthy condition, as has been demonstrated by a number of surgeons, it is probable that in the future surgical diseases of this organ will be more promptly recognized and treated accordingly. In leucocythemia, the splenomegaly being part of a general systemic condition, splenectomy is contraindicated.

Some Diagnostic Considerations with Regard to Splenomegaly.—More care than is usual should be taken in examining and outlining the spleen, and determining its size, by percussion and palpation. The enlarged spleen is sometimes mistaken for an enlarged left lobe of the liver, and, in cases where it extends down to the pelvis, for some pelvic tumor. If one can feel the edge or notch of the spleen, it is easy to determine an enlarged spleen, but sometimes it is impossible definitely to outline the organ. Pressing the organ forward and toward the median line may assist in bringing the anterior notched portion so that it may be palpated.

Dilating the colon with air would assist in determining the location of the kidney, which would lie posteriorly. The position of the kidney, especially if injected with colloidal silver, may be outlined with the *x*-ray. Omental tumors, as well as gastric carcinomas, may be mistaken for tumors of the spleen, as occurred in the experience of H. E. Giffin, of the Mayo Clinic. "While a slight enlargement of the spleen seems to occur without harm, it must also be remembered that splenic enlargement sometimes precedes the development of anemia by several years,—splenic anemia, and the spleen must be looked upon as a possible cause

of future trouble, especially if it be of considerable size. Its removal may then be considered." (Giffin.)

Tuberculous splenitis may occur without evidence of tuberculosis elsewhere. To exclude syphilis, a Wassermann reaction should be made in any case of doubt. An enlarged spleen frequently occurs with syphilitic cirrhosis of the liver. Congenital syphilis of the spleen is often found in children. Bush reports forty-two cases of cystic spleen. Carcinoma of the spleen is secondary and uncommon. The blood examinations are pathognomonic of lymphatic and myelogenous leucemia.

Splenic anemia, a disease of unknown origin, with progressive splenomegaly, an anemia of secondary type generally with leucopenia, some tendency to hematemesis, and a secondary cirrhosis of the liver, seems to be a definite entity.

Eighteen out of twenty-seven splenectomies at the Mayo Clinic were for splenic anemia. Dr. Louis B. Wilson summarizes the results of the pathological study of eighteen spleens removed from 1905 to 1912 at the Mayo Clinic, the clinical diagnosis being splenic anemia:

1. In each spleen there was hyperplasia of one or more of the constituent tissue elements. In two spleens the lymphoid tissue was so markedly overgrown as to suggest a diagnosis in one of lymphoma and in the other of lymphosarcoma. In three spleens the proliferation of the endothelium of the venous sinuses was most predominant. In thirteen spleens the process was a chronic diffuse one, involving all tissue elements.

2. While any case of primary splenomegaly may begin as an overgrowth of the lymphoid tissue or of the endothelium, in the ordinary course of events a secondary overgrowth of the stroma of the gland will later appear, accompanied by degeneration of the lymphoid or endothelial elements.

3. The largest spleens are those in which the lymphoid or endothelial hyperplasia is greatest. As the connective tissue overgrows, the spleen may be reduced in size, owing to reduction in the amount of lymphoid and endothelial elements.

4. The roughness of the exterior of the spleen is in direct proportion to the development of connective tissue within it.

5. In primary splenomegaly a secondary cirrhosis of the liver is associated with great connective tissue overgrowth and degeneration of the pulp of the spleen.

6. From what we know of hyperplasias in other organs, it would seem unnecessary to assume hypothetically the presence of three different causes for the production of the three primary histological types of splenic anemia.

7. The histopathological picture presented in all three types of spleens from cases of primary splenic anemia, seems to be in complete harmony with the hypothesis of the presence of a slowly acting local toxin.

8. There are certain spleens from clinically doubtful cases of splenic anemia which the pathologist cannot distinguish from the spleens from clinically undoubted cases. Yet it must be conceded that the findings of a great hyperplasia of one or all of the tissue elements of the spleen without other diagnostic lesions elsewhere in the body, is strong corroborative evidence of the clinical diagnosis of splenic anemia.

Dr. J. F. Bennie, of Kansas City, Mo., in a personal communication to the author, reports the following case:

Case 1.—Female, aged eighteen years; seen in country, November 29, 1910. History of appendicitis; eight weeks ago pain and tenderness over gall-bladder. Fever; vomiting at first; jaundice. Six weeks ago, tenderness left hypochondrium. Much abdominal distention; blood-count (doubtful accuracy) 8,000 whites, reds normal; 80 per cent hemoglobin; 80 per cent polymorphonuclears. Patient extremely pale. November 30, 1914. Liver enlarged, spleen more so. Tenderness over gall-bladder and spleen; moderate jaundice. Appetite poor, takes only liquid food; hemoglobin 40 per cent, whites 22,000 (poly. x); temperature at night to 102° F. December 8th. Red cells, 3,400,000. Stained smear showed all cells normal. January 8, 1911. Much better. Considerable pain over spleen and epigastrium. January 10, 1911. Laparotomy. Gall-bladder small, difficult to find. No stones; liver normal; pancreas enlarged. Spleen large and adherent to everything. Splenectomy with much difficulty. January 28th. Hemoglobin 90 per cent. Reds, $4,000,000; whites, 16,000. Increased number transitional; otherwise normal.

Pathological report of spleen: Interstitial splenitis. Patient was later reported well, married, and to have had a family.

Case 2.—(Dr. D. N. Eisendrath; personal letter). Diagnosis: perisplenitis, malarial hypertrophy of spleen; splenectomy; ileus from adhesions. Tailor, aged twenty-seven years, one year before he was seen, first noticed dull, continuous pain in the left flank, which would get quite severe five or six times a day. When patient walked this would radiate to the scrotum. The patient had several mild chills three or four months ago, no vomiting but slight nausea at times. Patient urinated very freely with a slight pain during act; amount and color seemed to be about normal. No history of jaundice. Patient lost twenty pounds in the last year. Had malaria when sixteen years of age; typhoid pneumonia when very young. Examination revealed heart and lungs negative. Abdomen: tender area below the left costal arch and it was thought that a small mass could be felt there. Cystoscopic examination revealed a large collar shaped prostate and a trabeculated bladder. The ureters could not be catheterized. X-ray examination of the kidneys and ureters was negative. Patient seemed to localize his pain entirely to the left costal arch from which it radiated downward. There was some tenderness here. The spleen itself could not be palpated. Abdomen otherwise negative.

On August 24, 1908, left rectus incision; abdomen opened; spleen found considerably larger than normal; capsule greatly thickened with a moderate number of adhesions, binding it to the stomach and to the under surface of the diaphragm. Gastrosplenic omentum attached over large surface of the spleen. There was an almond shaped yellowish area of perisplenitis. Kidney palpated through this incision and found negative. The case being diagnosed as one of chronic perisplenitis, removal of the spleen was decided upon.

Some weeks later, symptoms of intestinal obstruction developed and laparotomy was performed by Dr. L. L. McArthur. Dense adhesions were found and a volvulus of the small intestine well in the middle portion of the ileum, a twisting on the vertical axis of the intestine. The intestine involved in the volvulus seemed normal, but in the small triangle formed at the base of the mesentery by its twisting was found a kink of the small knuckle of the intestine, the lumen completely obliterated. Below this kink the intestine was collapsed, and above it dilated. Thrombus and knuckle of gut

easily reduced. Further exploration made and above to the left an old fibrous band incised and yet the stricture remained. Portions of the intestine just distal and proximal clamped with intestinal clamps and incision through gut walls, and anastomosis performed, using continuous waxed silk for suture material, first suture being a continuous Lembert before incision was made; second suture a continuous suture passing completely through intestinal wall uniting free edges. Patient alive.

Case 3.—(Dr. D. N. Eisendrath). Rupture of spleen. Patient, male, aged twenty-seven years, was admitted to the services of Dr. Andrews in the Cook County Hospital, Chicago, in December, 1906, with a diagnosis of crushing injury to the right hip. In the absence of Dr. Andrews I was asked to operate in the case. The constriction had been applied upon the thigh within five minutes after the accident, and the patrol officer said that but little blood had been lost. The patient met with the injury in the following manner. He was caught by the belting in a factory and carried upward to the ceiling. Examination of the thorax showed a fracture of the ninth and tenth ribs; a moderate amount of emphysema around it. I was attracted by the extreme pallor and almost pulseless condition of the patient. He was thirsty, and the ingestion of considerable quantities of liquid seemed to make but little difference in the condition of the pulse; the same was true of stimulation. Examination of the abdomen at this time was absolutely negative. There was no tenderness nor rigidity nor any evidence of free fluid. Patient was anesthetized, and an exploratory incision made over the left rectus on account of a possible injury of the spleen. Upon opening the peritoneal cavity, a considerable amount of free fluid escaped. Further examination showed there had been a laceration of the spleen, extending from the lower border up to the hilus and also cutting the lower border in two. It was quickly extirpated, and while this laparotomy was being performed the thigh was amputated. For a time after splenectomy the patient seemed to pick up, but the condition soon became worse again and he died about one-half hour later. No autopsy was performed, so it was impossible to state whether any other abdominal viscera were injured.

Dr. John H. Gibbon, of Philadelphia (by C. H. Musser, M. D.), published an interesting case of cyst

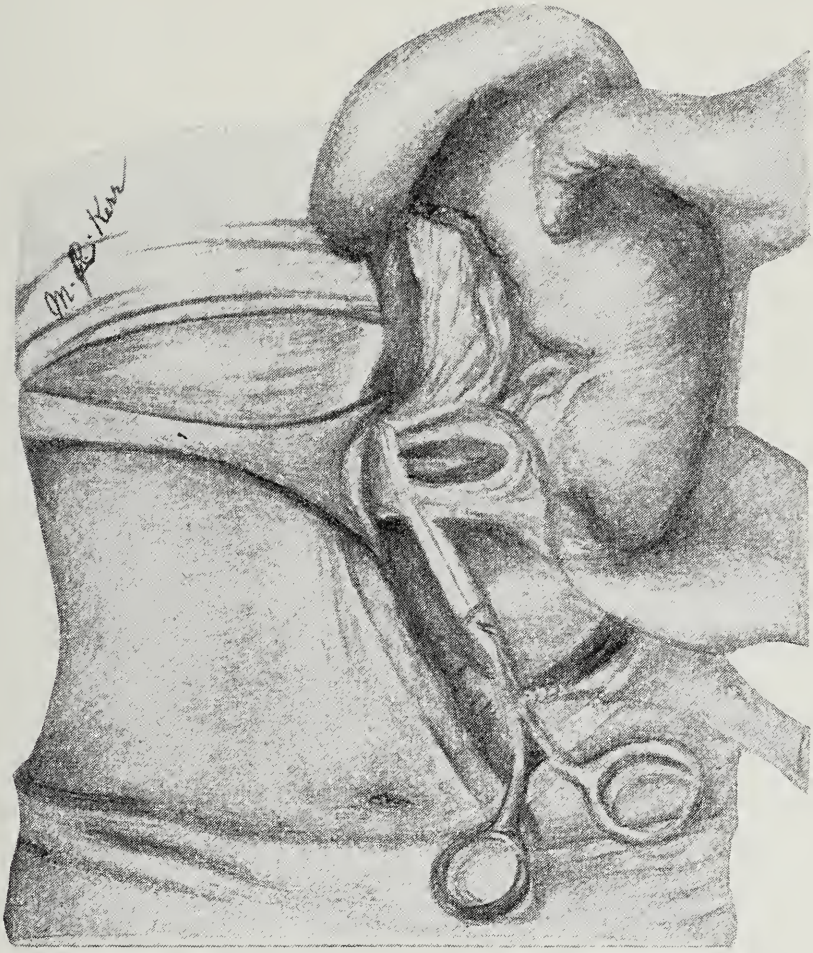

Fig.—Illustrating the method of applying the intestinal clamp in controlling the blood-supply of the spleen while performing splenectomy. (After W. J. Mayo.)

of the spleen. (*Am. Jour. of the Medical Sciences,* October, 1911, and a recent personal communication). The patient, an Irish woman aged twenty-five years, was rather anemic-looking. The abdomen showed the presence of a large tumor, most prominent in the epigastrium, and extending down to two inches above the anterior superior spine. The blood count was as follows: reds, 4,360,000; whites, 9,400; hemoglobin 70 per cent; the differential count showed polymorphonuclears, 65 per cent; lymphocytes, 29 per cent; mononuclears, 2 per cent; transitionals, 1 per cent; eosinophiles, 3 per cent. Doctor Gibbon, on opening the abdomen, found a large cyst adherent to the diaphragm and surrounded, except in the upper part, by splenic tissue.

TECHNIC

The technic of splenectomy is briefly as follows: Place the patient in an exaggerated Mayo-Robson position or marked lordosis. Make an incision through the left rectus, or Dr. A. D. Bevan's-shaped incision. Explore region of spleen; if adhesions are present, doubly ligate and cut between the adhesions to the omentum, the colon, and the abdominal wall. Adhesions to the diaphragm may give considerable trouble. If extensive, they may render the operation very dangerous. By the use of a large gauze pack the oozing can generally be controlled. The spleen is now free, except for its true pedicle. Deliver from the abdomen first the inferior pole and then the superior pole. When it is extremely large as in one of my cases (weighing fourteen and a half pounds), it is necessary for an assistant to support the organ after delivering it from the abdomen. Apply two intestinal clamps (covered with rubber tubing), one from below and, if convenient, one from above the pedicle. Insert interlocking sutures along the pedicle. Tie the splenic artery and

veins separately. Loosen the clamp slowly so as to be able to grasp promptly any branches that might still be oozing. As a further protection against hemorrhage, I put in a continuous running buttonhole stitch across the pedicle. This effectually prevented all oozing.

The tail of the pancreas approached close to the hilum of the spleen in one of my cases, and care was necessary to prevent grasping it in the pedicle clamp. "The splenic vessels may be reached, not only through the gastrosplenic, but through the gastro-hepatic or the gastrocolic omentum, or even from below upward through the transverse mesocolon." With sutures, close all the wounds made in the posterior peritoneum, and, if the gastrosplenic omentum was divided separately, suture its stump to the posterior abdominal wall. Close the abdomen. The accompanying illustration shows partially the method of clamping the pedicle of the spleen with intestinal clamp protected with rubber tubing.

Case 4.—(personal). Man, aged thirty-two years, single, laborer; native of Assyria; born about one hundred miles from Jerusalem. Patient was seen first in May, 1913, in consultation with Dr. A. N. Minear. Family history: father died about forty years of age of stomach (?) trouble; mother died at fifty-two years of age of intestinal obstruction (sick twenty-four hours). Patient came to America in 1907. Consulted Dr. A. N. Minear in 1912. Complained of weakness, anorexia, and pain in region of spleen. Was treated medically for about six months with no permanent relief. The blood examination showed an anemia and excluded leucocythemia. Splenectomy March, 1913.

An incision six inches long extending through the left rectus from a point four inches above to two inches below the level of the umbilicus was made; the abdomen opened, and the spleen exposed. The lower pole of the spleen was delivered, and then the upper pole was brought out through the incision.

The pedicle of the spleen was clamped with intestinal clamps protected with rubber tubing. A series of interlocking stitches were placed through the pedicle. The splenic artery and branches were ligated and the spleen was removed. A continuous chain stitch was inserted in the stump of the pedicle. The external incision was sutured in the usual manner.

The weight of the spleen was two pounds. Microscopically, the splenic tissue showed lymphoid hypertrophy, but no malignancy.

A blood-examination made November 11, 1914, about twenty months after operation, showed red-blood cells, 4,300,000; white-blood cells, 8,600; hemoglobin, 85 to 90 per cent. The patient says he feels well and is able to work regularly.

Case 5.—June 11, 1914, I was called to Idaho Falls, Idaho, to see a patient of Dr. William Kinnaird and Dr. S. S. Fuller in the latter's private hospital. The patient was a woman, aged thirty-three years, who was in a very critical condition. She had had an exploratory abdominal incision made a short time previously, and it was thought advisable not to attempt to remove the tumor from which she had been suffering. An examination showed a large tumor in the left side of the abdomen, extending from the ribs down to the pelvis. Careful palpation showed the tumor to have the characteristics of an enlarged spleen. As the patient's condition was gradually getting worse, a splenectomy was performed.

The technic was similar to that of the previous case, except that in this case there were extensive omental adhesions, which had to be doubly ligated and cut before it was possible to free the spleen. Owing to its extremely large size (weighing fourteen and a half pounds), an assistant had to support it while the bleeding was controlled. The tail of the pancreas extended to near the hilum of the spleen, so it was necessary to apply the pedicle clamps close to the body of the spleen in order to avoid injury to the pancreas.

The patient was in fairly good condition on leaving the operating room.

I did not have an opportunity to observe her condition, except for a few hours, as I had to leave for Salt Lake that evening. A later report from Dr. William Kinnaird showed that she did well for about one week, and then began to develop a high temperature. She finally lapsed into a semicomatose condition, which lasted till June 24, 1914, when she died. The autopsy showed no signs of infection in the abdominal cavity, but the pancreas was much enlarged and hard, and Doctor Kinnaird believed she had an acute pancreatitis. The pathological report was hyperplasia, round-cell infiltration, no tuberculosis, and no malignancy. (Doctor Schulte.)

The author desires to thank the following doctors for contributing interesting information in regard to the subject of splenectomy: Dr. W. J. Mayo, Rochester, Minn.; Dr. Allen B. Kanavel, Chicago; Dr. J. F. Bennie, Kansas City, Mo.; Dr. M. L. Harris, Chicago; Dr. Daniel Eisendrath, Chicago; Dr. John H. Gibbon, Philadelphia; Dr. Arthur Dean Bevan, Chicago; Dr. Norman Kerr, Chicago; Dr. A. N. Minear, Salt Lake, Utah.

GIANT-CELL TUMOR OF BONE

F. Gregory Connell, M. D.

OSHKOSH, WISCONSIN

A study of the giant-cell sarcomata of bone leads one directly into confusion, misunderstanding, and the use of words which apparently have different meanings to different people. The multiplicity of terms, such as medullary giant-cell sarcoma, myelogenous giant-cell sarcoma, myeloma, medullary giant-cell tumor, chronic (non-suppurative) hemorrhagic osteomyelitis, that have been used to describe such lesions, shows the indefiniteness of any one term. The distinction between these various tumors may be apparent theoretically; but, clinically and microscopically, it is not always present.

Adami[1] and Bland-Sutton[2] take these tumors away from the sarcomata and class them under a separate heading, the myelomata, which are described as tumors composed of tissue identical with that of normal red marrow of young bone. Bloodgood[3] realizing the disadvantage of the use of the term sarcoma, utilizes "tumor" as a substitute. Mallory[4] considers the giant cells merely an incident, and would not use the term giant-cell sarcoma. Barrie[5] claims that this class of tumor-formation is merely a type of inflammation, and suggests the term *chronic hemorrhagic osteomyelitis.*

That these tumors are usually benign, or comparatively non-malignant, was probably first recognized by König. Bergmann, Bramann, and Mikulicz, in 1895, were among the first to perform local or conservative

operations for such conditions, and cases treated upon this principle have frequently been reported since that time. The literature upon this subject may be found by referring to the writings of Bloodgood,[6] who, in 1902, was the first in America to treat such a case conservatively, and who in a series of excellent monographs, and annually in the December number of *Progressive Medicine,* has repeatedly and strenuously advocated local operation instead of amputation.

The relative benignancy of giant-celled tumors of bone would seem to have been conclusively demonstrated, yet a malignant type of this tumor does occur, as is shown by the report of Coley,[7] of the late J. C. Stewart,[8] and others. Stewart claims that even one fatal case is enough to disprove all theories of benignancy, and he asks the very pertinent question, "What is a giant-cell sarcoma?" The usual answer is, a sarcoma which contains a large number of giant cells, but just how many giant cells seems undetermined so long as they are enough to give a distinctive picture. Such an imperfect definition is of course unsatisfactory, and gives rise to confusion. It was claimed by Stewart that the matrix in which the giant cells lie is the essential element in this tumor, and is responsible for its clinical features; others arrive at this same conclusion. Jones and Whitman[9] say that "benignancy or malignancy must be decided for each case, independently, and the diagnosis reached on grounds entirely separate from the presence or absence of endothelial giant cells."

Another explanation for the conflicting opinions was presented by Mallory,[10] in 1911, in which he emphasizes the fact that there are two types of giant cells: a tumor giant cell and the other, a foreign body giant cell. (Fig. 1; see also Figs. 3 and 6.)

The former are usually large, clear, bladder-like

cells, with distinct outline, but staining faintly, within which there are multiple nuclei, or a large multilobulated nucleus, with mitotic figures, which stain deeply, and are situated in the center of the cell. These are usually not important features of the microscopic picture, although at times they may be numerous and conspicuous. They are true tumor-cells, result from multiple mitosis, and signify rapid growth.

The second type are, as a rule, smaller, with their cytoplasm fairly abundant, sharply defined, and staining deeply with acid dyes. The nuclei are smaller, uniform, more numerous without mitoses, and are often in clusters near the periphery of the cell. They resemble the osteoblast of the bone-marrow, and are not tumor-cells, but are merely a reaction to the presence of foreign bodies, and are due to the fusion of endothelial leucocytes. This form of origin has been well shown by Whitman.[11] They may occur in any part of the body, in tuberculosis or other granulomata, myoma of the uterus, cancer, in simple reparative or regenerative processes. They differ in no respect from the osteoblasts that occur in connection with bone under normal and pathological conditions. Foreign-body giant cells in a tumor involving bone usually signify only erosion and disintegration of bone. They may be present in either rapidly or slowly growing tumors, and do not signify rapid growth or malignancy. Both types may occur in the same specimen.

Mallory concludes that bone tumors should be classed according to their type cell, without reference to the presence or absence of giant cells, and that their malignancy should be determined by evidence of invasion of other tissue, their rapidity of growth, and by the number of mitotic figures present. According to Adam,[12] who does not agree with Mallory regarding these giant cells, Von Hansemann recognized three distinct forms of giant cells:

1. Foreign-body giant cell of endothelial and leucocytic origin.

2. Parenchymatous giant cell, tumor-cell proper, due to irregular mitosis, and lack of cell-division.

3. Myeloplaxes. These are present normally in the red marrow of bone as osteoblasts, and are the characteristic constituent of the myelomata.

The last differs from the first in that the nuclei are distributed evenly through the cell-body, and by an absence of central degeneration of the body, and differs from the second by the nuclei being well formed and of uniform size.

As long ago as 1897, McCosh[13] stated that in the future this group of bone-tumors would have to be subdivided.

Trotter,[14] in 1907, called attention to the necessity of a microscopic differentiation between the giant cells of bone-tumors. M. J. Stewart, of Leeds, England,[15] urged the necessity of differentiating between the true "myeloid" sarcoma and the malignant giant-cell sarcoma, and suggested that the histological diagnosis should be based upon the morphological characteristics of the giant cells, especially as regards their nuclei, claiming that in myeloid sarcoma they were numerous, uniform, small, and without mitosis, while in the malignant type they were few, irregular, often very large, and mitotic figures were frequent.

The unsettled condition regarding this pathological condition is again emphasized by the case in which Coley[16] reports an ossifying myositis, which subsequently became malignant, was diagnosed as giant-cell sarcoma by Professor Ewing, and in which amputation was followed by metastasis and death. Bloodgood[17] discusses this case, and, in speaking of the photomicrograph of the tissue, says it "does not look to me like a giant-cell sarcoma, but a very malignant

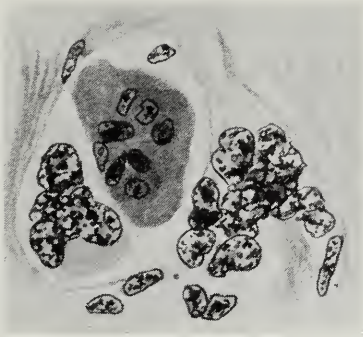

Fig. 1. Shows a foreign body giant cell between two tumor giant cells. From Mallory.

Fig. 2. A (lower right marked Fig. 10), from the tumors in the lung; B (upper right hand marked Fig. 8), from the secondary growth at shoulder; C (upper left hand marked Fig. 7), from the tumor removed at amputation. From Cathcart.

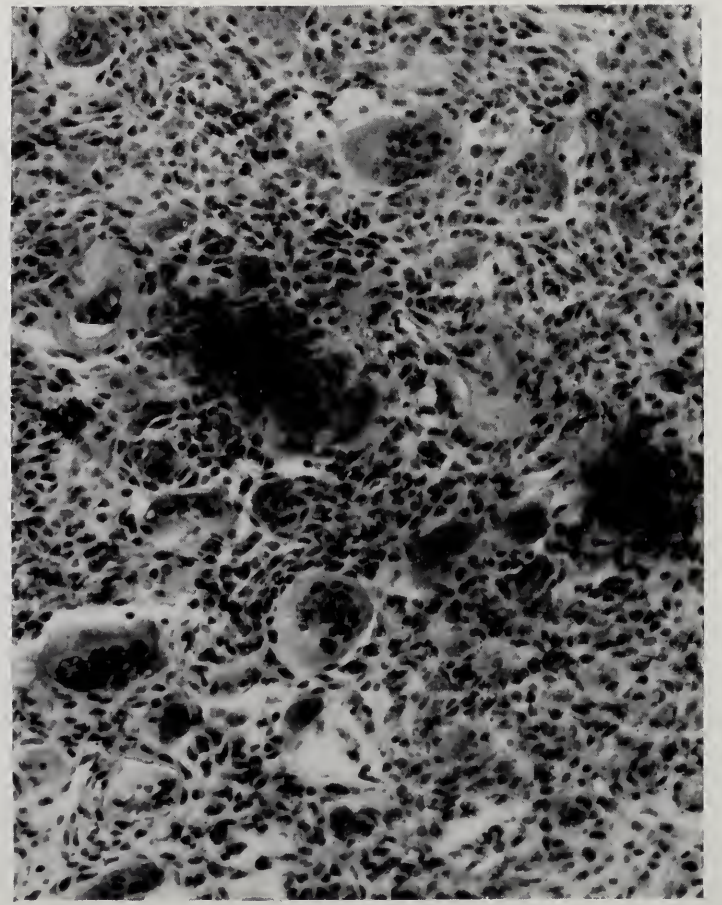

Fig. 3. Foreign body giant cells.[1]

Fig. 4. Case 2 before operation.

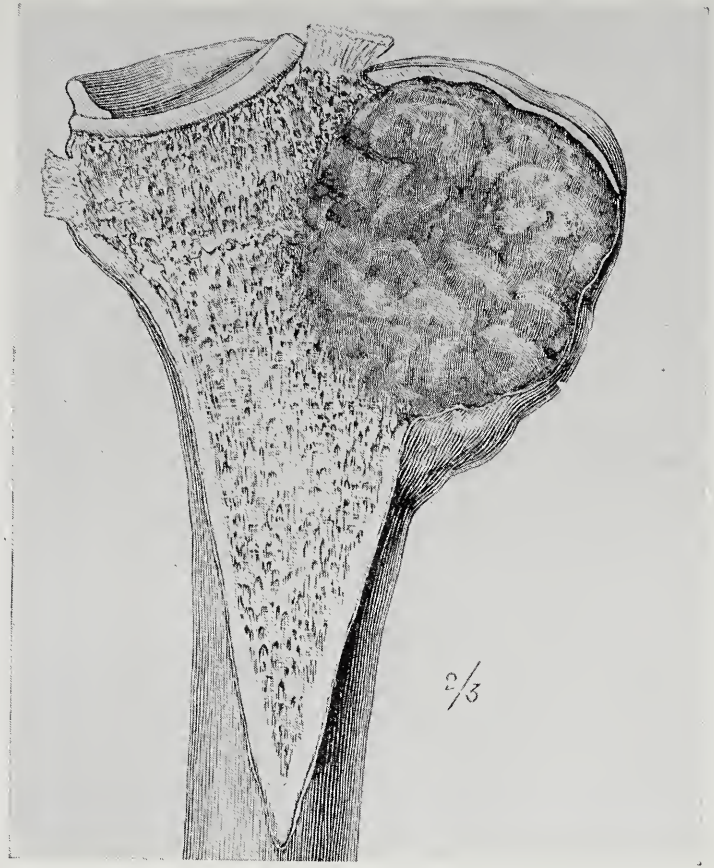

Fig. 5. Myeloma of head of tibia. From Sutton.

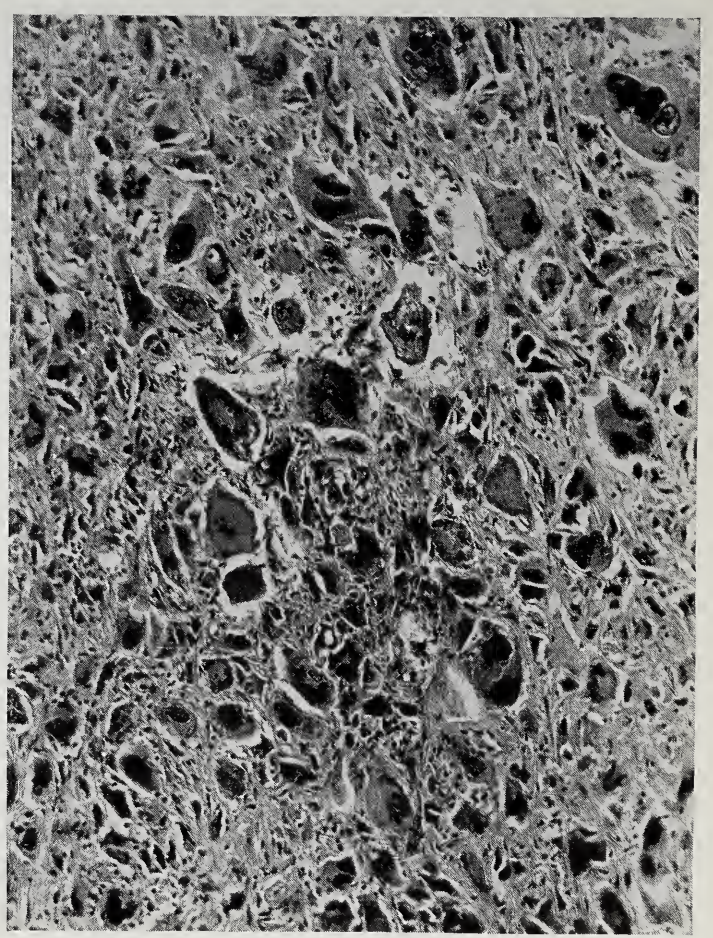

Fig. 6. Case 2, showing tumor giant cells. Compare with Fig. 3 (photomicrograph by Professor C. H. Bunting of the University of Wisconsin).

Fig. 7. Case 2, showing involvement of joint and femur.

Fig. 8. Case 2, longitudinal section of tumor after amputation. A, Granuloma removed at second operation.

mixed-cell sarcoma containing giant cells." Such circumstances would lead one to concur with Hertzler[18] when he says, "It must be recognized that there are no positive microscopic signs of sarcoma; it becomes necessary to resort to other evidence than that of the microscope, such as the history and macroscopic appearance of the growth."

Bloodgood[19] says, "Every now and then . . . I have been informed of a case of giant-cell sarcoma in which the patient died of metastasis. Some of these tumors I have been able to investigate and have found that the tumors were not giant-cell, but the most malignant sarcoma of the cellular type containing some giant cells, and that, when the metastatic tumors have been examined, there were no giant cells."

It is therefore interesting to refer to a case in Cathcart's work on "Innocent and Malignant Tumors,"[20] under the heading of malignant myeloid tumors. Case 2 is summarized as follows: "Fibroplastic tumor of the humerus containing myeloid cells; amputation at the shoulder-joint; recurrence of the disease; death three months after operation; secondary tumors in the lungs." The details of this case are given at length, but I shall quote only from that portion of the report that describes the tumors in the lung. He says that "the myeloid cells are not so numerous or as large as those in the larger tumor." Figure 2 is taken from Cathcart's book. A shows the myeloid cells in the lung; B, in the secondary growth in the shoulder; and C, from the tumor removed at amputation. The observations made upon this case by Mr. Mitchell Henry are doubly interesting when one considers the date upon which they were made; that is, October 20, 1858. With a change of some of the names mentioned, it might pass as a contemporary discussion on the subject. It once again shows the wisdom of the old say-

ing, "There is nothing new under the sun," and the marvelous facility of history to repeat itself. The observations upon this case are, without doubt, worthy of quotation at length.

The foregoing case is of great interest and importance from a surgical and pathological point of view, because it shows that the so-called myeloid tumor may run a course which cannot be distinguished from that of the most malignant cancer. The microscopic examination of the original tumor left no doubt as to the myeloid nature of the disease, and the surgeons present at the operation of amputation at the shoulder-joint expressed a confident opinion that the whole tumor had been removed together with the humerus to which it seemed to be exclusively confined. Notwithstanding this, in the course of eleven weeks a similar tumor grew from the scapula, and after the patient's death myeloid tumors were found in the lungs. The result of the case is disappointing to those who were inclined to accept the explanation that those quasi-malignant tumors of the extremities, in which amputation has formed a permanent cure, have in reality been examples of innocent kind of tumor, fittingly denominated "myeloid." Such tumors were first clearly described by Lebert, under the title of fibroplastic growths, and the characteristic myeloid cells were described and figured in his plates, although he does not seem to have been aware that the cells are similar in appearance to the polynucleated cells of the marrow and diploë of bones. The case just related, as well as the one detailed by Mr. Hutchinson in the last volume of the *Society's Transactions,* show that Mr. Henry Grey is in error in the conclusion to which he arrives in his paper on myeloid tumors in the *Medico-Chirurgical Transactions,* that they "are not malignant and when entirely removed never return"; indeed two of the cases narrated by Mr. Paget in the chapter in his "Lectures on Surgical Pathology," in which he first proposes the employment of the term "myeloid" would have seemed, *a priori,* to have thrown considerable doubt upon Mr. Grey's statement.

It is remarkable, however, that these myeloid cells should have been found in so many of the cases of amputation of limbs for supposed malignant disease, in which the operation is known to have been successful; but the question now arises whether they indicate anything more than that ossific changes are occurring in a tumor, in which case they will be found

indifferently in malignant and in innocent growths. Subsequent experience may enable us to determine the exact import of myeloid cells, but at present it seeems to me premature to elevate a characteristic which may, after all, be only accidental, into the test of a radical difference in the nature of a tumor.

Clinically, these tumors are usually located at the ends of long bones, most frequently at the upper end of the tibia. According to Bland-Sutton, they occur in this bone five times more frequently than in any other bone, and five times more frequently at its upper than in its lower extremity. They are more common in young people, and the same authority says that they are rare after twenty-five years, though Bloodgood reports a case at 66 years.

The onset is usually sudden, after a more or less severe traumatism. Tenderness is constant, but pain as a rule is not marked until there is a definite swelling. The course of the process is usually slow and of long duration. There may be crackling on palpation, the so-called "ping-pong" bone. There is no redness or dilated blood-vessels over the swelling.

The *x*-ray picture is not characteristic, and the earlier the case the more difficult is the differentiation between bone cyst, ostitis fibrosa cystica, and even metastasis of carcinoma or hypernephroma. In giant-celled sarcoma the expansion is more abrupt and circumscribed, and the distention greater, than in bone cyst or ostitis fibrosa, in which there is not so much expansion, the growth less localized, and more extensive longitudinally. In the benign growths, even with marked expansion, there is a preservation of bone-shell; while in the malignant medullary sarcoma the growth extends directly through the bone, without causing any marked preliminary distention. The *x*-ray is only an aid to diagnosis and should not be depended upon by itself in making a diagnosis.

On section the tumor is usually confined within the periosteum, the overlying bone may be very thin, the growth is definitely circumscribed, not infiltrating, and is easily removed from the bone-shell. The tumor proper is distinctly vascular, resembles young granulation issue, in which may be found whitish bodies varying in size from a pea to a grape seed, areas of osteitis fibrosa. The tissue in gross has been likened to red currant jelly, fresh cut liver, schmierkäse, and red barle-duc. In consistency it is friable, soft, and oozes, may be gelatinous, and might resemble section of brain tissue at recent autopsy. The microscopic picture varies greatly, giant cells of the osteoblast type predominating. Without these the histologic picture suggests granulation tissue. There are many endothelial-lined blood-spaces separated by connective-tissue stroma filled with spindle and round cells.

As has been said above, the diagnosis of giant-cell sarcoma is to a certain extent a matter of personal equation of the pathologist, as men of recognized ability may differ in the interpretation of such tissue.

Case 1.—Miss B. G., schoolgirl.[1] In 1907 this patient was referred to the late C. W. Oviatt by Dr. H. W. Abraham, of Appleton, Wis., because of a swelling of the upper jaw. The clinical picture was typical of tumor of the superior maxilla, the swelling was about the size of a baseball, with protrusion of the eyeball, and broadening of the nose, with egg-shell crackling on palpation. There was no evidence of disease of the mucous membrane of the mouth or of the alveolar process; there was no redness or edema of the skin. There was no pain or tenderness: her only complaint was a sense of fullness and the unsightliness of the deformity.

Under general anesthesia, the mucous membrane above the alveolar margin was incised, and the antrum was opened with a curette, and a spoonful of what appeared to be granulation tissue was removed for microscopic examination, and a piece of gauze was placed in the wound. Healing was prompt. The microscopic examination led to a diagnosis of giant-cell sarcoma (the giant cells being exclusively of the foreign-body

type), and excision of the superior maxilla was advised. (Fig. 3.)

Operation was refused, and the patient has received no medical attention. The swelling gradually subsided and she has enjoyed good health ever since. A recent report from Dr. Abraham says that the swelling has practically disappeared, and that the patient is apparently in the best of health.

It is well known, of course, that giant-celled epulis is a most benign tumor, and that recovery is to be expected after local or conservative treatment. However, this tumor was not an epulis, but a sarcoma of the antrum of Highmore, and the removal of a minute fragment for diagnosis would hardly be classed as even a local or conservative line of treatment. The case is of interest and value in emphasizing the reserve with which one should credit an occasional so-called cure following some more or less unusual line of treatment. The fact that spontaneous cure, or rather spontaneous subsidence of probable malignant growths, must be reckoned with is hereby emphasized.

That all cases of giant-cell sarcoma do not terminate as the above is well shown by Case 2.

Case 2.—Mrs. M. O'D., Russian; married; housewife; has lived in America nineteen months; was referred by Dr. W. J. Schmidt, of Mayville. Previous and personal histories negative; family history unknown. In October, 1913, patient first noticed a swelling, the size of a hazelnut, on the upper and inner aspect of the right tibia. There was complaint of swelling, redness, and tenderness, but no spontaneous pain. Treatment consisted of counter irritation, antirheumatic medicines, and later massage. The swelling increased and the pain became intense and constant so that in February, 1914, she could no longer walk, because of pain. A plaster cast was then applied to the knee, but with no improvement.

In May, 1914, the patient entered the hospital in the ambulance. The cast had been removed and the patient complained bitterly when the limb was moved. Examination revealed a thin, emaciated young woman, very apprehensive and crying out with pain on moving the limb or touching the swelling, which was about the size of an English walnut and

raised about half an inch from the surrounding surface at its highest point. It was located at the internal anterior aspect of the right tibial head, was distinctly localized, the skin over the swelling was freely movable, with no redness, edema, or dilated veins. There was no egg-shell crackling, or "ping-pong" bone.

The knee-joint was not swollen and appeared normal; active motion was not possible and passive motion caused severe pain. Because of the exquisite tenderness, the swelling was aspirated, but with negative results; temperature was normal, there was no leucocytosis; urine was negative, with Bence-Jones bodies absent.

X-ray picture, by Dr. C. H. Nims, showed a localized lobulated tumor in the head of the tibia (Fig. 4). Because of the marked similarity, I am reproducing a picture of a similar tumor from Bland-Sutton (Fig. 5).

On May 28, 1914, under ether, an elastic constrictor was applied and a longitudinal incision was made over the swelling. Division of the periosteum exposed the tumor which was distinctly localized and was composed of lobules of whitish gray cheesy material, with bony trabeculæ extending from the periphery toward the center.

This mass was easily removed with curette, and was about the size of a golf-ball. There was no distinct membrane lining the cavity which extended upward to the cartilage of the joint, posteriorly to the periosteum, and downward to the normal marrow of the medullary canal. It was separated from the opposite side of the head of the bone by a distinct wall. The walls of this cavity were curetted and cauterized with carbolic acid and alcohol.

Frozen sections by Dr. Emily Oberlin revealed a typical giant-cell sarcoma, the giant cells entirely predominating the histological picture, the true tumor giant cells and foreign body giant cells both being present (Fig. 6). The remaining cavity in the head of the bone was filled with subcutaneous fatty tissue, derived from the patient, the wound closed without drainage, and a plaster cast applied to the entire limb, the cast being cut at once.

Convalescence was noteworthy because of the constant and pitiful complaint of pain in the limb. There was primary union and the stitches were removed and the cast changed on the seventh day. On the twelfth day the fat escaped as an oily discharge from a minute opening at the upper angle of the wound. There were no local or general manifestations of

infection of the wound. This was not followed by any relief from the pain which was constantly complained of.

I expected that after the discharge of fat, the wound would close, and I went on my summer vacation and did not see the patient again until September 11, 1914. During the interval there had been an outgrowth of granulation tissue from the wound, which continually increased in size and was soon followed by swelling of the knee-joint; on July 9th an incision was made into the knee-joint, from which granulation tissue presented, which failed to close, and the granulating mass continually increased in size.

The patient was readmitted on September 11, 1914, with a granulating mass at the site of the previous incision and involving the knee-joint. This granuloma was about the size of an orange with a disintegrating crater in the center from which there came a very offensive discharge. The tissue seemed extremely sensitive and very vascular. There was no palpable enlargement of the iliac glands, and cachexia was absent. The patient had become a morphine habitué, and amputation was absolutely refused.

Wassermann, by Dr. Oberlin, negative. X-ray examination, at this time, showed destruction of head of the tibia, with invasion of the femur. (Fig. 7.) On September 17, 1914, under ether, this granuloma was excised. (Fig. 8-A.)

The entire head of the tibia was involved and the resultant cavity was cauterized with the actual cautery and loosely packed with gauze, and a trough splint applied.

Sections of tissue removed at this time showed some areas quite similar to that removed at the primary operation, while other areas showed mixed-cell sarcoma with fewer giant cells, and small round-cell infiltration. There was no amelioration of the symptoms following the cauterization, and on October 10, 1914, the cauterization was repeated under ether, and it was then noted that the growth had extended into the popliteal space. This second cauterization was followed by no change in the course of the disease. In spite of the suppurating mass, and the constant pain, there was an absence of the usual so-called "cachexia" and no regional infection. Permission to amputate was finally given, and on October 19, 1914, an aperiosteal amputation at the junction of the upper and middle thirds of the femur was performed. Figure 8 shows a longitudinal section of the tumor mass. Sections of this femur showed the same giant-cell formation. Amputation was followed by instantaneous cessation of pain, primary

union, most rapid convalescence and satisfactory immediate result.

CONCLUSIONS

1. A diagnosis of giant-cell sarcoma is not sufficient.

2. An attempt should be made to make a diagnosis, regardless of the presence or absence of giant cells.

3. The giant cells should be differentiated into either foreign-body, or tumor, giant cells.

4. Foreign-body, giant-cell tumors are more common and usually benign.

5. Tumor giant-cell growths are uncommon and less benign.

6. Giant cells have been found in pulmonary metastases.

7. The marked differences of opinion regarding these tumors may be due to a failure to differentiate between the giant cells.

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Note.—Since completing the above an important further contribution has been made by M. J. Stewart in The Lancet, Lond., 1914, November 28.

I am indebted to Professor C. H. Bunting for his interest in these tissues.

OVARIAN CARCINOMA IN A CHILD AGED ELEVEN

Van Buren Knott, M. D.

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Carcinoma of the ovary occurring at the early age of eleven is extremely unusual. The following brief history is of such a case. No effort has been made to include either a summary of the cancer problem in general or of the literature pertaining to the case in particular.

The patient, Gladys C., aged 11, a schoolgirl, had a negative family history. Her mother stated that she had always been an unusually healthy, vivacious child; had suffered from the ordinary diseases of childhood, but had never been seriously ill.

On April 3, 1910, she was brought to St. Joseph's Hospital by Dr. J. F. Taylor, who stated that he had first seen the child the day before, when she was complaining of severe abdominal pain not localized. This pain had been present for three days, before which time she had been feeling perfectly well. There was no menstrual history. Examination showed a well-nourished girl with rosy cheeks and unusually well developed for her age. Examination everywhere was negative with the exception of the abdomen, where the palpating hand at once encountered a large movable tumor, which rose from the pelvis to the level of the umbilicus. This tumor occupied the median line, but could be easily displaced to either side. It was quite tender on deep pressure and fluctuation could not

be elicited. When the tumor was displaced to the left, marked tenderness was found in the right inguinal region over the appendix, with rigidity of the right rectus.

The diagnosis was of tumor of the left ovary, with subacute appendicitis.

On April 4, 1910, operation was performed. Through a median incision, the tumor was at once exposed. It was found to consist of the left ovary, which together with the tube was easily removed, as it was at no place adherent. The appendix was found somewhat distended and acutely inflamed, and was removed. There was no free fluid within the peritoneal cavity, and the peritoneum everywhere was glossy and apparently normal. No lymphatic involvement or evidence of disease elsewhere within the cavity could be discovered.

Convalescence following the operation was entirely uneventful. From April until the following February, the child seemed well, had no pain, gained in weight and stature, and was noted for her happy, fun-loving disposition. Early in February she began to complain of vague abdominal pain, which was not at all constant. Soon her appetite which had been, as her mother said, "enormous," began to fail, until by the first of March she could scarcely be induced to take food. Dr. Taylor saw her about this time, but could, on physical examination, detect nothing pointing toward recurrence. She continued to lose weight and strength, and became very irritable. There was no constipation or vomiting. About April 1 some abdominal distention was noticed, which steadily increased. The pain by this time was becoming more constant and severe.

On April 19, she was again brought to the hospital. Examination revealed marked emaciation, an anxious,

drawn, facial expression, and marked abdominal distention, evidently due to a large amount of free fluid within the cavity. In short, it was the familiar picture of recurring malignant disease within the peritoneal cavity.

Operation was again performed on April 20, 1911. When the abdomen was opened through a median incision, a large amount of straw-colored fluid escaped. Scattered throughout the cavity were nodules involving parietal peritoneum, visceral peritoneum, intestine, and mesentery. These nodules were hard and irregular in outline. The ileum was adherent in many places, and at each point of adhesion there was a large nodular mass. The mesentery was filled with large nodular growths. The upper abdomen was involved as well, nodules being present in the liver and stomach. As relief was out of the question, a large mesenteric nodule was removed for examination, and the abdomen closed.

The child failed steadily following the second operation. The abdomen rapidly refilled with fluid, emaciation became more marked, pain required frequent administration of anodynes, and death occurred thirty-six days following the second operation. No necropsy was permitted.

The tumor was given to Drs. F. S. Johnson and W. G. Rowley of this city, who reported as follows:

The specimen is a large cystic tumor weighing three pounds, two ounces. It is ovoid in shape and nodular in contour, showing beneath the peritoneal surface many cysts varying in size from two and one-half inches in diameter down to microscopic size. The contents of these cysts are not uniform, and vary from a thin mucin-like fluid to a thick heavy yellow content that has proved to be degenerated, broken-down tissue. Attached to the tumor is a fallopian tube very much elongated and overdeveloped for an eleven-year-old girl. Accompanying the large tumor is an appendix from same subject as the large tumor. There are irregular nodular masses of tissue project-

ing above the surface of the tumor and into the cyst cavities. Sections made of appendix show no disturbance in the epithelium or the connective-tissue stroma. A round-cell infiltration with dilatation of tissue spaces showing inflammation, are the only departures from normal. Sections made of the fallopian tube show no pathologic deviation from normal. Sections made from many portions of the large tumor show a connective-tissue stroma rich in blood vessels and supporting areas of hyaline cartilage, groups of glia cells, strands of smooth muscle-fibers, tubules lined with stratified columnar cells, cyst-like spaces filled with mucin-like contents, and areas showing mucoid degeneration. Sections from one of the node-like projections into cyst space, show the columnar epithelium lining the tubules to be breaking through basement membrane and invading neighboring connective tissue and blood vessel walls. In one field, an artery of considerable size is plugged with proliferating epithelial cells. Diapedesis of redblood cells and infiltration with round cells is a noticeable feature in almost all sections examined.

Our diagnosis is teratoma in a state of carcinomatous degeneration.

The small nodule removed at the second operation was examined by Drs. Johnson and Rowley, who reported as follows:

On April 21, 1911, a small tumor one-half inch in diameter was handed us for microscopical examination and diagnosis. It was soft, friable, bloody, looking much like a blood-clot. Section showed it to consist of a stroma of connective tissue with a rich network of blood-vessels and supporting groups of columnar epithelial cells. Some were simply nests of proliferating columnar cells, while in other fields quite regular-looking glands lined with stratified columnar epithelium were noted. A most extensive diapedesis of red cells was everywhere to be seen. Diagnosis: carcinoma secondary to ovarian tumor, removed one year previously.

The one slide preserved from sections taken from the growth removed at the second operation has been examined by Dr. E. R. LeCount whose report follows:

The tumor is malignant and most like the proliferating cyst adenomas of the ovary. It belongs to the carcinoma group, and in these sections as in those from one or two similar

growths (they are very rare in young girls), also in young girls, I have found some changes or appearances indicating some small, but close, relationship to the so-called teratomatous growths.

The subsequent history of the case, together with the gross appearance of the abdominal contents at the second operation, when a condition of typical general abdominal carcinosis was found, coupled with the report of Drs. Johnson and Rowley concerning the microscopic diagnosis of the tissue removed at that time, would seem to justify the diagnosis of teratoma or embryoma of the ovary undergoing carcinomatous degeneration.

Added to this is the fact that the metastases took place by way of the lymphatics and were most extensive in distribution.

As a point of further interest it might be added that had the child not suffered an attack of acute appendicitis, the pain arising from which attracted the attention of her parents, this tumor would doubtless have much longer escaped detection.

DISCUSSION

Dr. A. W. Abbott (Minneapolis, Minn.): I do not think the doctor needs to apologize for the brevity of his paper, because in looking over quite a number of standard works, both in surgery and pathology, I find they say less about this subject than he does. In fact, most of our works pass over the subject with a very few lines, simply stating that it is known to exist.

In looking over an old work on surgery, which was perhaps the standard book at that time, 1871, my attention was arrested by a remark that shows the progress that has been made in pelvic surgery. The statement is that "Any of these tumors of the ovary, whether solid or malignant, should never be interfered with surgically!" Now, that, you see, was only forty-three years ago.

One of the peculiarities about these ovarian tumors in children is the multiplicity of forms which they may take. The ovary may have a tumor like those found in any other organ

of the body, or certain tumors which are peculiar to the ovary itself. Sutton, who made a pretty extensive research in this direction, says that the ovary is peculiar in this respect, that the malignant tumors, of the ovary, are more frequent in children than in adults, while, in the adult, the other parts of the body are more likely to be affected. Hecktone gives us about the same impression.

There have been some early cases of ovarian malignancy reported. Kelly reports one five years of age. Sutton says that they may occur very early in life, but not in the fetus, like sarcoma; but never before the end of the first year. The proportion of cases of cystadenomata to cancer of the ovary is given as one to five by Hecktone. This author also states, what I think is true, that the carcinoma of the ovary is apt to be double, much more so than in cancer in the breast, but that they do not usually occur synchronously, that is, one is formed and developed, and then later another one upon the other side.

Kelly states that tortions of the pedicle are much more frequent in children than in the adult. I was not aware of that.

I have not seen any dissections of this tumor, and of course cannot discuss that side of the question; but it would seem from the doctor's description that very probably this was of parovarian origin, for the reason that it was cystic, and also had the tube very much lengthened and stretched over the cyst.

I believe that one reason why we do not find carcinoma oftener in the ovaries is, that we do not examine all tumors thoroughly. We take out a piece here and there, and section it; but we find nothing but the ordinary cyst, perhaps a pappillary growth inside of the cyst, and we let it go at that. I have had one or two cases where there was an area of malignancy not larger than a pea in the tumor, just as we find elsewhere in beginning cancer.

In order to get at a satisfactory conclusion with regard to this point every tumor should be examined very critically; every portion of the tumor should be gone over before we can decide whether it is malignant or benign.

Dr. Arthur E. Hertzler (Kansas City, Mo.): This paper is so valuable that I would like to suggest for the sake of our tumor-nomenclature that the doctor change its title to "Teratoid Tumor." It is nearly always the endothelial cells of these tumors that blow out and become malignant.

Practically every one of this class of tumors is found in "Ewing's Study of the Mixed Tumor of the Testicle." This belongs to the same class, and you find one or two of them mentioned in my book on tumors.

Dr. C. D. Lockwood (Pasadena, Cal.): A year ago I reported a case of carcinoma, in a child of thirteen, secondary to ulcer, and at that time Dr. C. H. Mayo, discussing the paper, thought it was such an unusual thing to have carcinoma of the stomach in a child of thirteen that he wished the slides submitted to other authority. Dr. Black of Pasadena and Los Angeles had made the diagnosis. At his suggestion I submitted these slides to Dr. LeCount and Dr. Adamy. The former confirmed the diagnosis. He thought it was unnecessary to send the slides to Dr. Adamy.

I think, in this connection, it would be a good opportunity to report that this case was one of carcinoma of the stomach in a child of thirteen.

Dr. T. C. Witherspoon (Butte, Mont.): Just a word as to the origin of these tumors. The occurrence of the teratoma in the testicle and in the ovary is not uncommon. It is something we run across often.

It seems to me that some very valuable work has been done by Jaques Loeb in the direction of explaining why these tumors occur there. You remember his little work on artificial parthenogenesis in connection with the development of the female ovum, in the lower types of organism, either by pricking the ovum or changing it by chemical or thermic means. He has thus been able to increase the nourishment of that ovum until it develops at least to a certain stage.

The reason that he gives is, that the normal surface tension of the ovum is so great or is of such a character that it prohibits normal nutrition, and therefore division and growth of the cells cannot occur. In other words, the ovum is simply pent up and held in statu quo for the time being until perforated by the sperm cell, or until its permeability is increased by mechanic or chemic means, then it grows.

Now, it would be quite possible that these germinal cells in both the testicle and the ovary might under some conditions be altered so as to proliferate, and develop these tumors.

We can think of a real parthenogenesis, and not an impaction (Verneuil). It is simply a matter of increased nutrition

to the ovum cell, and then a rapid multiplication of cells with differentiation into both connective tissue and epithelium.

The epitheleal cells or ectodermic cells frequently become malignant in that they manifest a marked tendency to infiltrate.

Dr. Knott (closing): I have very little to say in closing. One reason for bringing this case before the Association was the fact that it is extremely unusual to find, at least in my own experience, carcinoma occurring at so early an age.

We generally recognize carcinoma as a disease of old age, rather than of youth. So far as the teratomata are concerned we originally considered them as benign tumors. It has been demonstrated in many cases other than the one mentioned today that the teratoma, while a benign tumor, probably at some early stage of its occurrence, is one most prone to take on malignant degeneration. In other words, there have been teratomata reported in which sarcomatous or carcinomatous degeneration has taken place, and taken place very early, and because of the fact that this carcinomatous degeneration occurred at so early an age as eleven years, it seems to me unusual.

CAVERNOUS AND CYSTIC LYMPHANGIOMA OF THE CECUM

WITH REPORT OF A CASE

EDMUND A. BABLER, M. D., F. A. C. S.

ST. LOUIS, MISSOURI

If Wegner's position relative to the congenital origin of lymphangioma is correct, it is not at all surprising that my review of the available literature has failed to show the record of a case of lymphangioma of the cecum or colon. With the present-day method of microscopic examination of all tumors removed at operation, surgeons have become appreciative of the importance of the method and the frequency of tumors heretofore regarded as of infrequent occurrence. It is not at all improbable that the future will show that lymphangioma of the large intestine is not so uncommon as the literature now indicates.

Wegner[1] classifies lymphangioma into (1) simple, (2) cystic, and (3) cavernous. In our specimen, Professor Welch found both cystic and cavernous forms of lymphangioma.

It is conceded by some writers that inflammatory changes may occur in these growths, and that the latter are detected in a large percentage of the cases before the patient attains the thirtieth year. In twenty-five of the fifty-two cases of lymphangioma of various parts of the body collected by Klemm,[2] the patient was under 25, while in thirty-eight cases the patient was under 40. Eleven were over 40 years of age.

Sudden elevation of temperature in cases of lymphangiomas is perhaps explained by inflammatory changes therein or by the intra-abdominal rupture of one or more of the cysts. Many of the cysts contain a cloudy substance. Kuttner[3] has discussed inflammation and pus-formation occurring in lymphangioma. It is somewhat difficult to explain the inflammatory changes which have so frequently been noted in these growths. If they are of congenital origin why should inflammatory changes occur therein?

Hagenbach[4] has reported a very interesting case of symmetrical lymphangioma of the neck which was noted at birth. Thiebierge and Gottheil[5] have each reported a case of lymphangiomas of the skin, while various authors have reported lymphangioma of the mesentery, small intestine, fallopian tubes, diaphragm, etc.

The following very interesting case of cystic and cavernous lymphangioma (Welch) of the cecum is of especial interest to the surgeon in view of the fact that the clinical picture simulated so closely that of an acute infection of the appendix. It is also of interest owing to the unusual location of the growth.

REPORT OF CASE

History.—W. B., a lad aged 15, was admitted to the medical service of the St. Louis City Hospital, June 28, 1913, and on the following day the patient was transferred to my surgical service. The patient stated that previous to onset of the present illness he had enjoyed good health. His father had died from consumption and his mother and one sister were afflicted with the disease at the present time. The present illness began four days before with sudden severe pain in the region of the appendix. The pain was followed by vomiting and fever. Examination showed a pulse of 116, a temperature of 102, and a leukocyte count of 16,000. Examination showed the patient's expression somewhat anxious, the abdomen slightly distended in the region of the appendix, and considerable tenderness and rigidity in the region of the appendix.

There was no enlargement of the inguinal glands. The left side of the abdomen did not show rigidity. Rectal examination failed to show a mass. A distinct mass, however, could be outlined about an inch above and an inch and a half anterior to the superior spine of the ilium. The patient's right limb was retracted. The patient presented all the symptoms and signs of an acute infection of the appendix with abscess formation.

Operation.—Immediate operation was performed. When the abdomen was opened through the usual incision along the border of the rectus muscle, a firm mass about the size of a man's fist was found situated to the outer side of and intimately connected with the cecum. The appendix was free and did not show evidence of inflammation. The mesentery glands were not enlarged. The growth was almost entirely covered with peritoneum. The cecum did not show infiltration or inflammatory change. There was no evidence of tuberculosis.

The diagnosis of probable carcinoma of the cecum was made, and the cecum with the entire ascending colon and about eight inches of the ileum were excised, and a side-to-side anastomosis by means of a Murphy button was made. The button was expelled on the seventh day, and the patient made an uninterrupted recovery. The patient was discharged, July 10. He is alive and in apparent perfect health at this time.

Dr. F. B. Hall made a röntgenogram of the colon and terminal loop of the ileum, showing the present condition of the anastomosis.

PATHOLOGIC REPORT BY PROFESSOR WELCH

The specimen consists of a portion of the intestine with a tumor attached to the intestinal wall, hardened in alcohol. The intestine is the lower end of the ileum, 14 cm. in length, and the cecum 9 cm. in length, with the appendix. Partly enveloping the cecum and growing from its outer wall is a tumor measuring approximately 10 cm. in length (parallel with the axis of the gut), 6 cm. in thickness and 5 cm. in breadth. The tumor is, except over a part of the area, invested with peritoneum. The shape is irregularly oval with a somewhat irregular, nodular outer smooth surface.

Upon section the tumor presents a variegated appearance and is seen to be composed of a solid part, apparently connective and adipose tissue, and of many larger and smaller cysts and channels. The cysts vary from those just visible

to the naked eye to those 2 cm. in diameter. From some the contents have escaped, others contain clear gelatinous material, and others blood either fresh or altered in color. The walls of the cysts in general are thin and smooth, but some show projections and suggest fusion of adjacent cavities.

The solid tissue is in places hemorrhagic, in other places grayish and firm, or with the appearance of fat.

The tumor is intimately incorporated with the outer wall of the cecum, from which it cannot be detached. It has not invaded the mucous membrane, which is intact.

Microscopic Examination.—The cystic and canalicular spaces are seen to be dilated lymphatic vessels lined by endothelial cells. The contents vary, being sometimes, especially in the smaller spaces, homogeneous, and in others containing fibrin, leukocytes and red corpuscles in varying proportions. The leukocytes are mainly lymphocytes, and some of the lymph cavities are completely filled with these cells. Other cavities contain only or chiefly red corpuscles. The walls of the cysts and dilated vessels show bundles and detached strands of smooth muscle, often much hypertrophied.

The tissue between the cavernous spaces and channels is fibrillated connective tissue, or, in places, adipose tissue. It is partly doubtless the pre-existing tissue, but much of it is of new formation. In places it is delicately fibrillated and edematous; in other places more densely fibrous. A notable feature is the presence of smooth muscular cells, often hypertrophied, not only in the walls of the lymphatic and blood vessels, but also scattered in the intervening tissue. Here are also fusiform and stellate fibroblasts.

Accumulations of lymphoid cells are present not only within the cysts and channels, but also in their walls and likewise in the intervening stroma. There are more or less extensive extravasations of blood, mostly fresh, both within the lymph-spaces and in the tissues.

The cystic dilatations are in places so numerous as to give a honeycombed appearance to the sections; in other areas the tissue is more solid, but throughout the new growth such dilatations exist. Sections which include the junction of the tumor and the cecum show that similar, although narrower, dilatations of the lymph vessels occur in the submucous, muscular and subserous coats.

There is proliferation of the endothelium of the peritoneum, and considerable new growth of connective tissue and cellular infiltration in the serous membrane immediately adjacent

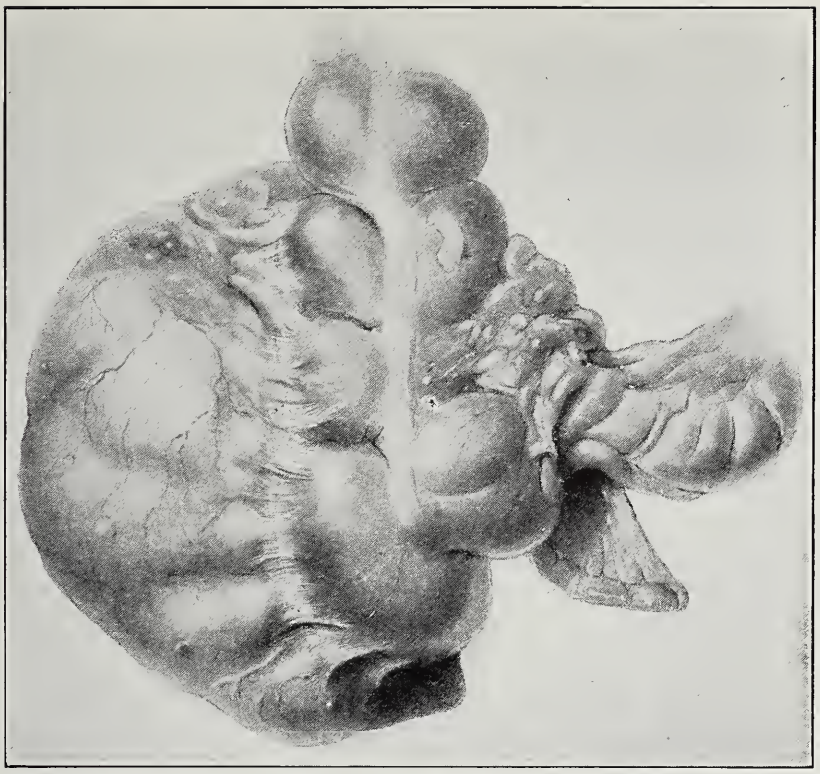

Fig. 1.—Anterior view of growth showing intimate relationship to cecum.

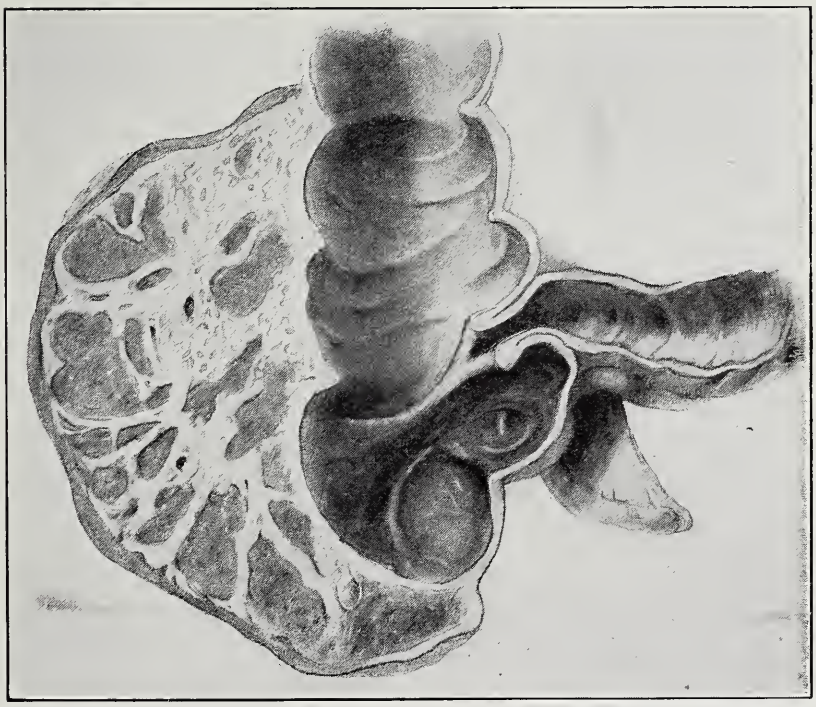

Fig. 2.—Longitudinal section of tumor and cecum.

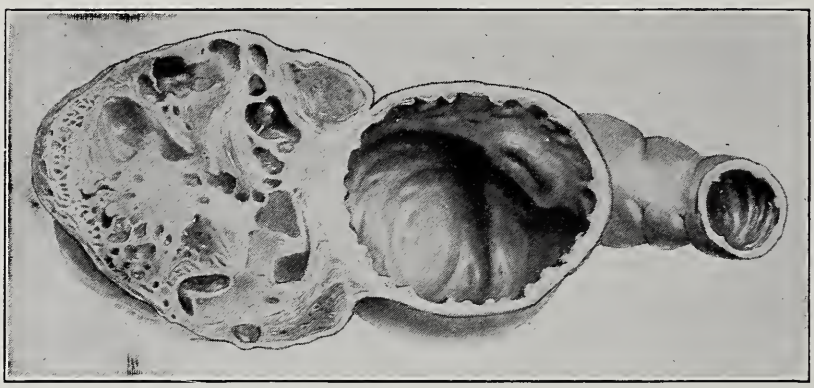

Fig. 3.—Transverse section of specimen.

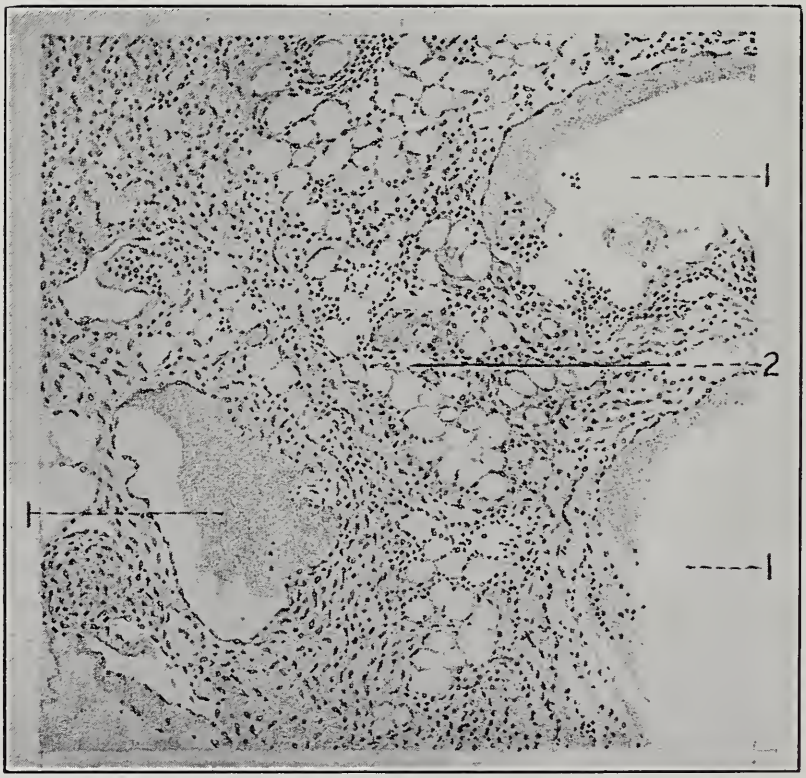

Fig. 4.—1, Lymph spaces, surrounded by edematous connective tissue (2), and infiltrating fat.

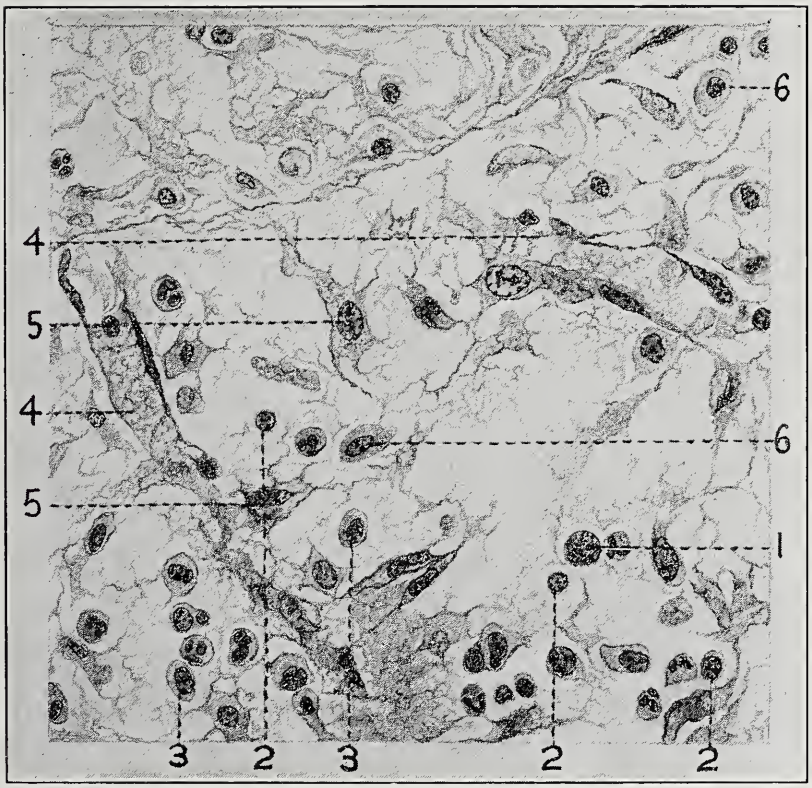

FIG. 5.—High-power picture, showing character of connective-tissue stroma. 1, Eosinophil cell; 2, lymphocytes; 3, plasma cells; 4, newly formed capillaries; 5, fibroblasts; 6, large mononuclears.

to the tumor. The lymphatic vessels, frequently dilated, are filled with lymphocytes.

The same colloid or coagulated material present in the lumina of the lymph cysts can sometimes be recognized in the meshes of the tissue composing the solid part of the tumor. Fibrillated fibrin is also present in some places in the tissue.

Diagnosis.—Cystic cavernous lymphangioma.

Remarks.—To what extent, if any, there is an actual new formation of lymphatic vessels it is difficult to say. It is possible that all are pre-existing or these with lymphatics formed in the new tissue. The fibrous tissue and muscular cells between the lymphatics are partly of new formation, and perhaps the tumor might be regarded as a fibromyolymphangioma, but it seems unnecessary to interpret the growth as other than composed essentially of dilated lymphatic vessels accompanied, as is usual in lymphangiomas, with some new formation of connective tissue and accumulation of lymphoid cells. New growth of smooth muscle is common in lymphangiomas, although in the present instance the development of the smooth muscle cells at a distance from the lymphatic vascular walls is a notable feature.

The most plausible explanation of this type of lymphangioma refers it to some error in congenital development.

There seems to be no active infectious inflammation, although chronic inflammatory changes are present. As regards the general interpretation of lymphangiomas as inflammatory in origin, that is another matter which relates to the general theories of this form of neoplasm. I am personally more inclined to the hypothesis of congenital origin for neoplasms of the type in this case.

There are extensive extravasations of blood, both fresh and old, but I do not regard these as essential features so far as the diagnosis of the primary condition is concerned.

Shortly after I received the foregoing report, Professor Welch wrote me as follows:

I have examined further sections from a piece which I cut from the tumor. There is nothing significant to add. The formation of masses of lymphatic tissue, growing in a polypoid form into the lumina of some of the cysts, was more noticeable, but I think that I spoke of such lymphatic tissue in my report to you. This speaks for new formation of lymphatic vessels, but is not conclusive. In fact, in many cases it is impossible to say whether the cysts are from pre-

existing lymphatics or from newly formed ones, and this you will find discussed in most of the articles on the subject.

The new sections show partial organization of the coagulated contents in some of the cystic dilatations. The hypertrophied muscle in the walls of the cysts and channels is conspicuous, as also are dilated blood vessels. Of course, there is a good deal of chronic inflammatory change, as I have already noted.

COMMENT

The case of lymphangioma herein reported is of especial interest to the abdominal surgeon because of its close proximity to the appendix and the infrequency of its occurrence in the colon. Hildebrand[6] has expressed the opinion that lymphangiomas are sometimes diagnosed as lipoma or fibroma.

The fact that a tumor of congenital origin may become so intimately connected to the cecum and be subjected to certain changes which manifest themselves by high leukocyte count, severe abdominal pain, vomiting, high temperature and, in fact, the usual clinical manifestations of an infected appendix, is worthy of careful consideration. Hertzler[7] and other writers have expressed the belief that these tumors are of inflammatory origin.

Note.—I am especially indebted to Dr. W. W. Grant for his interest in the case; also to Professor Opie and Drs. Thomas and Tiedemann of Washington University, as well as to Dr. Hertzler of Kansas City, for valuable assistance. My indebtedness to Professor Welch of Johns Hopkins is evidenced by his pathologic report.

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DISCUSSION

Dr. Arthur E. Hertzler (Kansas City, Mo.): You may recall that in October, 1913, I published a study of "Lymphan-

gioma" in Surgery, Gynecology, and Obstetrics. At that time I collected the literature on this subject, as far as I knew, completely, and when I got this specimen from Dr. Babler I renewed the search of the literature, without revealing anything additional.

There is no other case, as Dr. Babler has stated, involving the large gut which has been reported. There is one small one in the small intestine reported by Sick.

The only question now is as to the genesis of this tumor. Schmidt has reported one; and, I think, there are one or two others reported which developed about gastric ulcers.

The question, then, is the relation of this case to inflammatory changes. You know in the tongue the lymphangiomas blow up, get thick, subside, lie dormant for a time, and inflame again and again until a large part of the tongue is involved.

Now the question is whether this tumor is formed from multiplication of the lymphatics *per se,* or whether it is surrounding tissue-changes which produce the change within the lymphatics.

I have some slides here, from other studies, notably degenerations in myomatous tumors. I show here a low-power photomicrograph of a mid portion of the tumor. The long cells shown are non-striated muscle fibers. I will show also a higher-powered picture in a moment.

I now show in the illustration very faintly the hypertrophied blood-vessel. You can see fairly well the light membrane with the endothelial lining destroyed, and with the blood-clot within its lumen.

This illustration shows the dividing walls between the cysts filled with a coagulum. You will see a number of small vessels likewise with thickened vessel-walls, with the hypertrophied connective tissue in between, and then here again the coagulated lymph. Here you will see also very well-preserved red blood corpuscles.

Here you see the lymph-channel. This is a low-power, forty-eight millimeter lens. This septum here contains many hypertrophied non-striated muscle cells, and is really the crux of the situation, which I will explain later on.

Here is a blood-vessel from a degenerating uterus. You see here the enormous hypertrophy of the vessel walls, and here the degenerating connective tissue about it. You will recognize here the cells. They look very similar to the collection that Dr. Babler has shown. It is generally supposed

that these newly formed channels are distinguished by lack of protoplasm; but that is not so, as you will see there.

Here you will see numerous sinuses, likewise lined with endothelium. These are complete sinuses, as you will recognize from the relation to each other, very much like lymph sinuses in the specimen shown.

The question is as to the genesis of these tumors. In the specimen I showed of the hypertrophied blood-vessel, and in the specimen of Dr. Babler, there has been some antecedent change outside of the lymph-vessels. Therefore it is not a simple hypertrophy of lymphatic tissue but that the surrounding tissue has reacted with it. As Dr. Babler stated, the connective tissue between these cysts shows much recent and past inflammation, so that we know that this tumor is relatively old.

Now, the tumor itself is sub-peritoneal. The peritoneum of the gut encloses this tumor. The surface of peritoneum covering the tumor shows a reactive process and an inflammatory exudate of considerable magnitude. The catastrophe that brought the boy to the operation no doubt is a recent hemorrhagic infarction in the mesentery quite outside of the tumor area. The hemorrhagic exudate showed blood-clots sufficient to explain the pain.

What probably occurred in this case, so far as we know now, is, that a lymphatic process once started, whether existing as congenital, as is probable, or whether a lymphatic hyperplasia, the result of some antecedent inflammation, as in the case following an ulcer of the stomach, we do not know. The probabilities are that the tumor was actually derived from an embryonic anlage, because of the amount of muscle tissue about the lymph sinuses. We could hardly account for that increase of muscle-tissue on the theory that it is a stasis of the lymphatics with the subsequent dilatation and the subsequent reaction. Therefore we must conclude that this is actually a congenital tumor, in which secondary reaction has taken place, and is therefore, strictly speaking, neoplastic formation, and not the result of reactive processes.

Dr. Emil H. Beckman (Rochester, Minn.): Clinically, we see instances of lymphangioma or lymphoceles which are probably of the same origin as this specimen.

In the neck, for instance, it is not unusual to find a symmetrical tumor, usually round, which lies in the anterior jugular region, and seems to arise from the anterior jugular

group of lymphatics, which consist of a single sac, as a rule, and can be enucleated. In addition to the single sac, a single large sac and two or three smaller sacs may occasionally be found, which seem to connect intimately with the chain of lymphatics.

It would seem that the main lymph-channel was blocked, and the lymph itself accumulated in the lymph-gland which could not empty.

I remember one case in particular in which a large tumor lay in the axilla, completely filling it so that it interfered with the action of the arm. The tumor was soft, but not pulsating, and was supposed to be a lipoma. On making an incision down to it, however, it was found to be an enormous lymphangioma with large sacs, one of them nearly four inches in diameter. The enucleation of the mass made a complete dissection of the entire axilla.

The lymphangiomas I have seen have been lying along a lymph-channel, and I wonder if in this case it might not be possible that this was the chain of lymphatics which ran behind the cecum in the peritoneal folds. Whether congenital or not, it had become enlarged and pushed around to the side forming this tumor.

REPORT OF THE CHAIRMAN OF THE FIRST GENERAL STUDY TOUR OF THE WESTERN SURGICAL ASSOCIATION

James F. Percy, M. D., F. A. C. S., Secretary

GALESBURY, ILLINOIS

At the last meeting of our Association at St. Louis, President Jackson, in his address, suggested the advisability of our Association planning a clinical surgical tour every alternate year, after the plan followed by the Clinical Surgical Society for so many years. A motion was made that the incoming President, Dr. Davis, appoint a committee to arrange the details for such a prospective trip. President Davis appointed Allen B. Kanavel, D. N. Eisendrath, and J. F. Percy a committee on organization. This committee met in Chicago. Dr. Eisendrath was made Secretary, and Dr. Percy, Chairman. Correspondence was at once commenced with all the members of the Society and the following finally agreed to become members of the party:

Dr. A. E. Benjamin, Minneapolis, Minn.
Dr. F. G. Connell, Oshkosh, Wis.
Dr. Edward Evans, La Crosse, Wis.
Dr. Leonard Freeman, Denver, Colo.
Dr. J. C. Hancock, Dubuque, Iowa
Dr. Jabez N. Jackson, Kansas City, Mo.
Dr. A. F. Jonas, Omaha, Neb.
Dr. Donald MacRae, Council Bluffs, Iowa

Dr. Arthur T. Mann, Minneapolis, Minn.
Dr. J. F. Percy, Galesburg, Ill.
Dr. E. P. Quain, Bismarck, N. D.
Dr. H. M. Richter, Chicago, Ill.

The following members joined our party in London at the meeting of the Clinical Congress:

Dr. Allen B. Kanavel, Chicago, Ill.
Dr. J. Rilus Eastman, Indianapolis, Ind.
Dr. H. B. Sweetser, Minneapolis, Minn.

Drs. Arnold Schwyzer and Gustave Schwyzer of St. Paul, Minn., joined us, first, in Zurich and, later, in London.

Thus, eighteen of the twenty members who signified a willingness to go when the matter was brought up at the St. Louis meeting, did go. By the time it became known how many of our party would make the tour, the subject was taken up with a tourist agency, which would look after the details of the business management. But it was found to be too late to arrange an itinerary of clinics on the other side. In addition to this, steamship accommodations were already scarce. At this juncture, through the Tourist Agency of New York, we learned that the Georgia Surgeons' Club, under the direction of Dr. R. M. Harbin, of Rome, Georgia, as Secretary, had already prepared a program which included most of the better known European clinics. A telegram was received from Dr. Harbin, asking us to join the Georgia Surgeons' Club. After consulting with the two other members of the Committee, and with those of our Association who had expressed a desire to make the trip, the invitation was accepted. Your Committee learned from Dr. Harbin that the total membership of his party was sixteen. This, with the eighteen from our Association, promised to make just the right number, both from the standpoint of congeniality and availability

for attendance at the clinics. Your Committee was surprised—and this was also true of the members of the Association who had joined the Tour—when it was discovered that the Tourist Company had booked, under the spacious name of "American Surgeons' Clinical Tour to the Clinical Congress of Surgeons of North America" every one who had applied to them for membership in the Tour. This brought the membership practically up to two hundred persons. In this number were included the wives and children of some of the members. Those of the Western Surgical Association who were accompanied by their wives were Dr. Leonard Freeman, Dr. Jabez N. Jackson (wife and daughter), Dr. A. F. Jonas, Dr. Donald MacRae, Dr. Arthur T. Mann, and Dr. J. F. Percy.

Our party left New York, Saturday, June 13, 1914, on the White Star Line Steamer "Oceanic." We arrived in Cherbourg, June 20, after an uneventful but delightful voyage, during which a number of papers were read by volunteers from among the surgeons of the party. Among these was a lecture by Dr. Jabez N. Jackson on the "Surgical Conscience." Another was given by Dr. A. F. Jonas on the "Duty of Technic," and one by Dr. Leonard Freeman on "Post-Operative Duty." Dr. F. G. Connell discussed "Landmarks for Borderline Cases"; Dr. J. F. Percy, "Borderland of Insanity." Dr. O. O. Cooper, of Hinton, West Virginia, and a member of the Georgia Surgeons' Club, gave a very delightful lecture on "Some Great Paintings Surgeons Should See."

Going over on the boat, in order to visit the Clinics most effectively, it was decided that our party should be grouped into sections. This was made necessary because it was known that in most of the clinics we should visit the seating capacity was small. The one hundred thirty physicians in the party were divided

into five groups. Four of the groups made an assessment of three dollars for each member, while the fifth made an assessment of five dollars. The writer was made Treasurer for all the groups. This money was used for various incidental expenses that were found not to be covered by the money paid the Tourist Agency. At the present time, as Treasurer of the Company, I hold a balance of $150.75.[1] One of the purposes of this fund was its use for some suitable souvenir, which should be presented to those who not only gave us clinics but extended to us other courtesies. The war developing as it did, before our party had an opportunity to get together in the person of its Executive Committee, has left this part of our work uncompleted. It is a question now, because of the exigencies brought about by the war, whether this part of our program can be carried out. Dr. Harbin and the writer are at present corresponding with the hope that we can devise some form of recognizing those who added so much to the pleasure and profit of our visit. Our difficulty is to learn accurately how many of these men, valuable members of the European medical profession, have lost their lives in this war.

Even should your patience permit, it would be impossible to give in a report of this character every detail of what we gained in the way of impressions and knowledge of the work of our European confrères. We can mention only the most impressive of our experiences. In passing, we can say, and we think without fear of contradiction, that the net result of our observations in all of the places visited, is, that European surgery and medicine are much like

[1]It was later voted by the Executive Committee to turn this balance in the treasury over to Dr. F. F. Simpson, Pittsburgh, Pa., Treasurer of American Physicians, for the Aid of Belgian Medical Men. This was done March 24, 1915.

our own. With this statement is included the work of our English brethren.

The American surgeon who has never personally viewed the work in Europe, with its splendid system of preliminary general and scientific medical education, expects to find the work of the highest degree, because it is always supposed to be based upon the best that this education affords. In America we deplore our inadequate preliminary scientific medical education, and have for years, and well we may. But, judged from what we saw in our hasty view of European medicine, their "perfect" system of medical education and preliminary training before entering the actual practice of medicine, has not protected the European public to any greater degree than is true in our own country.

From what we observed in practically every city we visited, European educational methods have not altogether protected the suffering public, either from the competent or incompetent knave or the deluded fool in the practice of both medicine and surgery. Expressing a purely personal opinion, in contrasting the European system of government supervision with our own of absolute non-interference upon the part of any central authority, ours is of more value to the public than theirs. On the Continent and in England we saw inferior, or at least irresponsible, men retained in responsible positions regardless of the indifferent character of their work.

One of the best known surgeons, upon finding a gastric ulcer which had not been diagnosed previous to opening the abdomen, remarked that "the Germans could not diagnose gastric ulcer previous to operation as easily as did the English and American surgeons."

In what I have previously written, I realize that I have violated all the rules of legitimate criticism by

not relating first the really impressive and good things that were ours to enjoy while on the Tour we are attempting so briefly to describe. I may be pardoned if I relate two things that are always impressed upon the American surgeon visiting Europe for the first time. The first is the inferior position of their nurses in relation to the care of the sick, and, as a consequence, the lack of care of patients, as we understand it, from the nursing standpoint, in this country. The nurses we saw were more or less slovenly,—hair unkempt, caps awry, uniforms unbecoming. Most of them, in looks and actions, reminded one of the ward maids often found in many of our American hospitals. Most of the nurses get very fat; and it is said that one can tell by their size how long they have been in training.

The second very impressive fact, even in the most noted and best clinics, is their failure to give what we would consider a good or even a safe anesthetic. The whole subject of this phase of surgery seems to be tabooed everywhere in Europe. In an European clinic the writer has never seen an anesthetist who seemed happy while administering either chloroform or ether. Again, I have never seen a patient under a general anesthetic for whom I did not feel some anxiety, and take a deep breath of relief when the drug was withdrawn at the completion of the operation. This experience with general anesthetics on the part of the European surgeon must account for the very general use of so-called local anesthesia. The use of a local anesthetic in gastro-enterostomies and in resections of the stomach, as I and others have seen in patients who were in good shape, especially, for ether, is to make use of what is an anesthetic in name only, for this type of operation. Many of our party remarked that patients in Europe tolerated more than

would the average of our people. The exposure to air and water, the cyanosis from the drug administered, the pulling on the chin, the exposure of the body, and the tapping on the cornea with the finger, the administration of drugs by hypodermic—all reminded one of anesthesias in our own country twenty years ago.

Possibly it is to be regretted that the time-limit on papers prevents a fuller description of what our party was privileged to see and learn while abroad. As it is, we can hope to give only a very limited running impression of what we saw and heard.

Exclusive of England, Ireland, and Scotland, our party had the choice of fifty-four clinics by some of the best known surgeons of Europe. In addition to this, we were given three notable receptions. The first of these, by the Paris Academy of Medicine, was a remarkable tribute to American surgeons by the profession of Paris. The affair consisted of a reception at the Academy, headed by the venerable President of the University of France. In addition, we were given a very high-class vaudeville performance in the faculty room, and a superb buffet luncheon. We were told that this was the first time in the history of the Academy that either a play or food had been introduced into the building for any purpose. The faculty room was a wonderful place in which to meet, not only because of its architecture, but because its walls were covered with one of the most valuable collections of tapestries in the world. The clinics in Paris, which we were privileged to attend, were of a very high order, and made upon our party a lasting impression for good. A number queried why American surgeons did not visit these clinics at least as frequently as they do those of Germany. I think the chief reason for this, as contrasted with Berlin or Vienna, even though the Paris surgeons are doing

some wonderful work, is the lack of organization. Then, too, the clinical services are not systematized, so that the great abundance of material is not utilized to the best advantage. Then one usually gets the impression that the average French surgeon is covered with a sort of shell, which it takes some little acquaintance and persistence to get under. This once accomplished, they are a most affable and instructive group of men.

An unfortunate circumstance attended the close of our visit. Some of our party, abroad for the first time, gave interviews to reporters of the *Paris Herald,* in which some of the things unfamiliar to American operating-rooms were criticised rather freely. Among the things noticed was the wearing of beards by the operators, and the failure to remove anything but their coats when operating,—i. e., their sterile gowns were placed over their street clothes, not even the collar or tie being removed. Their hospitals and laboratories were also rather freely commented upon. From a newspaper standpoint, especially when little was published in the way of commendation of Paris medicine and surgery, this was probably considered good stuff; but from the viewpoint of guests criticising, in a rather bald sort of way, those who had entertained them, it was most unfortunate. The Paris profession came back at their critics in a dignified and illuminating way.

The advice of your Committee is, that, if another Tour of this character be contemplated by our Association, all newspaper interviews should be intrusted to a member of the party who has the judgment, experience, and courtesy sufficient not to break the rules of observant international good sense.

I regret again that we cannot mention by name and

describe the character of individual work that we witnessed in some of the clinics of this great city.

Our second notable reception was by Professor Sauerbruch in Zurich, notable in the clinic that he had prepared for us, and in the chartering of a large steamer on Lake Zurich. This latter was not only a most wonderfully delightful exhibition of international good-will, but it was unique in the number and variety of "stunts" pulled off for our entertainment. As long as this is a private report to this Association, I am sure it will do no harm to state that by common consent of the members of our party, this clinic, more than any other visited, gave great evidence from its Chief, his assistants, and nurses, of being controlled by a master whose work made an impression of finish that is seldom seen. His lung work, which he showed us in all its varied phases, left little to be desired, both as to the technic of the operation itself and as to the living and cured patients, whom he exhibited in large numbers. His relationship to his patients, and his evident interest and concern to do just the right thing for them as a surgeon and a man, were most refreshing. In addition to this, Professor Sauerbruch has a most delightful and companionable personality.

At the end of this paper I shall add the names of the cities included in our itinerary, and the dates we visited them. This will permit me to jump, in a descriptive way, to Jena as the third place in our journey, where we were entertained both socially and scientifically by another master of surgery, Professor Lexer. This clinic in a new and modern hospital showed also the impress of its master on every hand. Lexer seems to believe it wise, at least so he said, "never to let anybody do anything that you can do yourself." In fact, he told us as much during a de-

scription of his work in the clinic. His reason was that this was the only way that you could get things done in the way you wanted them. This was told in the presence of his assistants. How they felt about it, their impassive German faces gave no sign while their chief was talking. The manipulation of tissues, especially of joints, and his varied methods of doing plastics, were most instructive and valuable. They showed a patience and skill that has so far not been true of any of our prominent American surgeons.

In the evening, Professor and Mrs. Lexer gave a most delightful entertainment. It was in the square of the town of Jena. This square was used as a market-place in the day time. Tables, covered with red and white cloths, were loaded with good things to eat and to drink. We were also entertained by music and song. Our party was surrounded by a rather high, movable, white picket-fence. This kept the populace from disturbing us, but not from seeing our enjoyment of the whole delightful affair.

We were also entertained, in a rich way, by the immortal Professor Kocher, of Berne. He opened his home and his beautiful gardens, and gave us a clinic in a way that will remain a pleasant memory of a great man as long as any member of our party shall have mind enough left to appreciate the work of the best that has been really accomplished in the surgery of the world.

We were also lavishly entertained by Professor Witzel at Duesseldorf. He operated on one case for us, an appendix, in order to illustrate his method of assuring perfect asepsis. He also did an amputation of a leg to show his method of hemostasis.

It is not necessary to mention either Vienna or Berlin by name, as these places are so well known to

all medical men, whether they have had the privilege of visiting them or not.

It will not be amiss to speak of some of the things to be avoided should our Association attempt another similar excursion across the Atlantic. In the first place, our party was too large to be handled efficiently, either in the clinics or the hotels. The only advantage was that we had private trains throughout our Tour on the other side. Too much was left to the Tourist Agency that took us over. We did not always get the first-class accommodations for which we paid. The profession in Europe is not financially able to entertain large numbers; the expense is too great, and cannot be looked for, or accepted, by those who make these tours in the future. There should be no promiscuous advertising, inviting everybody to come and join the Tour, as was done in this instance. A party of thirty-five should be the maximum. This will insure a private car on any of the European railroads, and is all that is required for convenience and comfort. In addition, a small party can be accommodated in one hotel. This adds greatly to the convenience and enjoyment of the Tour. A small party can insure, in a measure at least, the character and standing of those who are to join it. No one should join a party of this character except upon invitation. Only in this way can be avoided disagreeable and humiliating experiences during the trip, and the leaving of bad impressions on the minds of the professional men in the various countries visited.

This Tour was peculiarly fortunate in having in the company so many representative men of our Association. Dr. Jackson, Dr. Jonas, and Dr. Freeman were particularly helpful when it came to deciding questions that concerned all of the party. Dr. Jonas gave a lot of his time, willingly and cheerfully, in trans-

lating into English from the German and the French, where our language was not spoken by the surgeon giving the clinic. Had he and Dr. Freeman and Dr. Quain not helped us out in this way so many times, much that we did get from these visits to the clinics would have been denied us.

Last, but by no means least, our good Secretary, Dr. Mann, with his ability to organize, and his inherent knowledge of what to do in order to get the most possible out of our visit, came to our aid whenever and wherever he found it possible to be of real help. I would seriously recommend that, if this Association ever undertakes another trip of this character, it try and prevail upon these men, whose names I have mentioned, to accompany it, because of their knowledge of languages and traveling experience.

In closing, I would be glad to devote some time to describing the Clinical Congress which at the time was the inspiration and the objective point of our Tour. It was a big affair. The brain that first conceived it has placed American Medicine and Surgery under an everlasting debt of gratitude. It has given us a reputation and a standing throughout Europe that makes for mutual respect and appreciation of our work over here, and the aims and aspirations that are back of it.

Those of us who saw Europe and England last summer will not see the same countries again. Should we ever be privileged to return there, all will be different. Shot and shell, the emissaries of frenzied men, will not only have changed the attitude toward life and all its activities held by the average common fellow of all those war-torn countries, but it will have altered the life-outlook of the high and the low, of every man, woman, and child. Is it without the bounds of reason, does it violate the laws of human

probability and experience, to say that the medical men of the present generation in Europe will likewise have been beaten back to where the science of medicine—and indeed all science—is but a mirage, and that honorable endeavor along the old lines which they so far advanced, is a thing for which they will no longer really care?

If the holocaust, which has spread like a great pall over all of them, engulfing their business, their arts, and their science, also destroys or renders inactive any one of a dozen physicians and surgeons whom we could name, working in general or special lines, the work of the world in our profession will be put back irreparably for such a number of years that no human mind can estimate.

If, as Professor Czerny, of Heidelberg, said recently in an editorial in one of his journals, the German government is putting some of her best medical men on the firing-line as common soldiers, not only Europe, but America, will feel a loss that no words of mine can adequately describe. If Europe, out of the battles which are weakening and robbing it of its chief glory as a scientific Mecca towards which we have been glad to turn, loses this because of the destruction of some of her best men, may our profession in America do its utmost to keep the lamp of scientific endeavor burning in a way, if needs be, to teach the children of men that science did not die when Europe invited the Gods of War to destroy it.

Program of Clinics visited by the members of the Western Surgical Association in connection with the American Surgeons' Clinical Tour to the Clinical Congress of Surgeons of North America:

Baltimore, June 8, 1914.
Philadelphia, June 9, 1914.
New York, June 10, 1914.

Saturday, June 13, 1914, sailed on White Star Line, Steamer "Oceanic" for Cherbourg, France. Arrived at that port, June 20, 1914.

Paris, June 20-24.
Berne, Switzerland, June 26.
Zurich, June 27-28.
Munich, June 30, July 1.
Vienna, July 3-4.
Dresden, July 7.
Berlin, July 9-11.
Leipsig, July 12-13.
Jena, July 14.
Frankfort, a. M., July 15-16.
Heidelberg, July 17.
Dusseldorf, July 20.
Cologne, July 21.
Brussels, July 22.
London, July 27 to August 3.

A PLEA FOR HIGHER HOSPITAL EFFICIENCY AND STANDARDIZATION

Horace G. Wetherill, M. D., F. A. C. S.

DENVER, COLORADO

Every human endeavor may become vastly more effective if those who are employed in the same or in similar occupations can be brought to see the wisdom of coöperating with each other and coördinating the work they are doing.

Friendly competition is in no particular incompatible with such coöperation. The interchange of ideas and experiences, and the combination of forces for the attainment of common ends, are, as a rule, mutually helpful. The most modern and successful scientific surgery is peculiarly dependent upon efficient teamwork. Everything connected with it, from a diagnosis for a particular patient to the compilation of the ultimate results, calls for the intelligent and skillful coöperation of many individuals. No single surgeon, however capable and skillful he may be, can hope for the highest success without the direct or indirect aid of his fellows in his own and other fields, nor is good surgical work possible without a well-equipped hospital in which to work. A hospital in which the surgeon may find, and collaborate with, trained assistants and laboratory-workers, is now acknowledged to be indispensable.

My present purpose is to point out that in common practice this kind of coöperation has not gone far enough; that the highest efficiency in medical and sur-

gical work, and the best results attainable, may be reached only after there has been a union formed between all of the hospitals in any given community for the promotion of the common good. The most perfect organization of any particular hospital creates merely a perfect unit, while the greatest good to the greatest number of hospitals, physicians, and patients, can come only from a combination of all, for the good of all. That such coöperation among the hospitals of any community could be made the means through which the efficiency and economy of their administration would be promoted is certain. Through a comparison of methods, a discussion of ways and means, and a standardization of practice in many particulars, simplicity and economy and better results would come.

That the advantages of collective bargaining as applied to hospital supplies, food, and furnishings alone would more than repay such coöperation, has been shown by the New York Bureau of Standards and Supplies, through which many of the large eastern hospitals are provided at jobbers' prices with those things they require. What we, as surgeons, are more directly interested in, however, is the benefit that could be made to accrue to the cause of better surgery and better surgeons. The welfare and well-being of all surgical patients could be promoted, and the results of surgery, and the credit and standing of surgeons and hospitals alike, could be improved.

In my address as President of the Colorado State Medical Association, in 1906, this matter was referred to; and the suggestion was made that a Board of Hospital Councillors be created by appointing two or three medical representatives from each hospital in a city to form such a council. Many important matters might properly come before such a composite body for consideration. Some of these questions are related

to certain hospital abuses with which you are all familiar. They are peculiar to no single locality, and characteristic of no particular hospital. It is humiliating to confess that they are more or less general; and it is undeniably evident that nothing short of municipal, state, and national hospital coöperation and standardization will eliminate them.

You will have anticipated that I refer to the total inadequacy of preparation of many of those who aspire to do, and who actually do, major surgery under the protection of our open hospitals; to those whose commercial and surgical aspirations are so interwoven that any surgical judgment they might otherwise possess is overmastered by the mania for operating, and the consideration of the share of the fee to be paid to the operator; and, worst of all, to those few who use the hospitals to cover the criminal practices of their offices, or the offices of others with whom they are associated.

Open hospitals, governed and controlled by laymen, in the management of which the medical and surgical staff, if one exists, has little or no voice, are everywhere in this country guilty of one or more of these offenses.

One occasionally hears whispered about among doctors or nurses the details of some surgical atrocity committed by some untrained or incompetent operator who should not be admitted to practice surgery under the protection and implied guaranty of any hospital. Without skill, experience, or training for a sufficient period under a competent surgeon, and without sufficient knowledge of surgical pathology or of surgical diagnosis, he is allowed to send patients into many hospitals, and, unquestioned and unrestrained, he attempts the most delicate and difficult operations, hop-

ing that he may be able, by the grace of God, to get away with it. The results you all know.

Even with all the advantages of the best trained and most skillful mind and hand, and with the last word in modern scientific surgery fresh in his memory, the most capable surgeon, with the highest and best of motives and a conservatism which considers nothing but the welfare of the patient, feels his limitations, and fears failure. Fools rush in where angels fear to tread, and nothing short of a general medical board of control for all the hospitals in every community, with full authority to say who may and who may not operate in such hospitals, will ever protect the hospitals and the people against such operators, or protect such operators against themselves.

Some one has well said that "the tragedy of today is not the tragedy of the criminal, but of the incompetent, and not of the absolutely incompetent, but of the relatively incompetent. It is the tragedy of the man who has the best intentions and the best character and a fair equipment for his work, but who has not a thorough equipment, and who cannot do the thing he starts to do in the best possible way. Society is crowded with half-equipped workers, with men and women who are honest and earnest and not incapable, but who are not up to the level of the very best work."

When surgical ambitions are associated in an individual with the necessary recklessness and indifference to the welfare of the patient and a longing for at least half of the fee to be got by operating, the worst is yet to come—for the patient. Surgical conscientiousness, diagnostic skill based upon an adequate knowledge of surgical pathology, a conservatism based on faith in the resisting and recuperative powers of the human economy, and a knowledge when not to

operate and not to use food and drugs, are better qualifications for a surgeon than the nicest dexterity and technical skill of the most spectacular operator on earth.

Among other desirable ends to be attained by coördination, is the adoption of a uniform system of hospital accounting and statistics. The Philadelphia County Medical Society appointed a Committee on Hospital Efficiency, which made a most interesting and valuable report to the Society at its meeting, October 21, 1914, in which they recommend that the County Medical Society take the following steps with a view to accomplishing this purpose:

1. Prepare a uniform system of morbidity records and statistics based on the International Classification of Diseases and Conditions, and urge every institution to adopt the system with such variations as may be suggested by the needs of individual hospitals.
2. Urge each hospital to perfect its system of medical records so that they will constitute an adequate foundation for the proposed follow-up system.
3. Urge each hospital to keep an accurate record of the condition of patients at the time of their discharge, and to provide for an accurate follow-up of patients after their discharge.
4. Urge each hospital to put in operation a definite plan for obtaining the information desired in regard to each discharged patient in one of the following ways: (a) by writing at stated intervals to the patient, his physician, or one of his friends who has a permanent address; (b) by sending a representative of the hospital, at stated intervals, to interview the patient or his physician; (c) by taking whatever steps may be necessary to insure the return of the patient to the hospital at the proper time for further examination, and, in some cases, for treatment.

Regarding such higher hospital efficiency the Committee of the Philadelphia County Medical Society further reports:

Your Committee believes that it would be of great assistance in paving the way for the proposed local study if some

organization with adequate resources at its command would first make a preliminary study of the entire hospital situation in the United States, and would define standards by which to measure the efficiency of a hospital. Such a report would provide your Committee and the efficiency committee in each hospital in Philadelphia with a working plan for accomplishing the purpose for which they were appointed.

We, therefore, recommend that the County Medical Society endorse the request which the American Medical Association, the American Congress of Clinical Surgeons, and the American Hospital Association have forwarded to the Carnegie Foundation, asking that the Foundation prepare a report on the classification and standardization of hospitals—a report that will perform as great a service for the hospitals of this country as the "Report on Medical Education" has already performed for the medical schools.

The promotion of hospital efficiency and standardization, the elevation of hospital morals, and the exclusion of the unfit and the incompetent from hospital practice, have now become a matter of paramount necessity because of the importance of the hospital in the present scheme of medical education. The hospital is now deemed as essential to the two last years of modern medical teaching as the laboratories are to the first two years, and here also the coördination of these two promotes the efficiency of both.

Dr. William Osler, in his address, "The Hospital as a College," delivered before the New York Academy of Medicine in 1903, says:

The work of an institute in which there is no teaching is rarely first-class. There is not that keen interest, nor the thorough study of the cases, nor amid the exigencies of the busy life is the hospital physician able to escape clinical slovenliness unless he teaches and in turn is taught by assistants and students. It is, I think, safe to say that in a hospital with students in the wards, the patients are more carefully looked after, their diseases more fully studied and fewer mistakes are made. . . . The objection often raised that patients do not like to have students in the wards is entirely fancied. In my experience it is just the reverse.

Though it may seem to be somewhat inconsistent with my strictures and criticism of American hospital management in some of its phases, I desire to pay a tribute to the splendid achievements of modern scientific surgery and the general excellence of surgical results everywhere in this country, even in many small communities. I am sure we all believe, however, that these results could be improved and would be better if such evils as are here referred to could be eliminated through an efficient system of coördination and standardization of our hospitals. So also, the individual surgeon would become a better trained and safer surgeon if he were obliged to qualify for admission to hospital practice by meeting certain standardized surgical requirements before such privileges were accorded him. Notwithstanding the ultimate success and high attainments of many self-made surgeons, the general average could be vastly improved by better hospital organization in the same way that the present requirements for a medical degree have improved the output of the medical schools, through the demand for higher educational requirements and better preliminary education for admission.

We must, as a matter of course, appreciate the fact that the regulation and standardization of the hospitals of this country would present even greater difficulties than were confronted in the standardization of the medical schools; but these difficulties are not insuperable, and may be overcome in time if the attempts of the American College of Surgeons and other allied surgical societies are endorsed and sustained as they should be.

In support of the efforts of the American Medical Association, the American Congress of Clinical Surgeons, the American College of Surgeons, and the Philadelphia County Medical Society, may I be per-

mitted to suggest the propriety of the Western Surgical Association giving this movement consideration and such support as a representative committee of this Association may see fit to recommend?

DISCUSSION

Dr. E. P. Quain (Bismarck, N. D.): It was a great pleasure to listen to the president's paper this morning on the standardization of surgery, and an equal pleasure to hear now this paper on the standardization of the hospital.

This question has been very near to me for a great many years; and the more I have thought about it, the more have I become convinced that here is the root of an evil against which we have been fighting so long. The poor and often dishonest surgery carried on in many of the hospitals which claim recognition because they are "open" to the profession, is appalling. In its widest sense an "open" hospital invites anyone who has, or claims to have, an M. D. degree, to bring his patients and operate according to his ambition, surgical or otherwise, without responsibility or worry as to bad results. On the other hand, a hospital board which takes upon itself the duty of choosing a staff of well-qualified men who are held responsible for all the results of treatment in the hospital, has taken a step in the direction of standardization of hospitals and of surgery.

From an observation of this kind of hospital management I am convinced that we cannot urge too strongly upon all hospital boards the necessity of keeping their doors closed, not only against medical and surgical imposters, but against every practitioner until he is properly investigated and vouched for by the staff.

Dr. Wetherill referred to the American Hospital Association. I happen to know that the question of the "open or closed" hospital question has been discussed in this Association for the past two or three years. I think if we would use our influence upon members of this Association to get them to think about these matters, and to work along these lines for the next few years, a great deal of good might be accomplished.

I know of a superintendent of nurses who for several years had charge of a typical "open" hospital in the East, and who later served in a hospital where the more "closed" methods I have referred to were in practice. She was a very religious

woman; and when the question of returning to the "open" hospital practice came up, she promptly stated that her conscience forbade her to do so. She preferred to leave her position rather than be a party to the administration of medical treatment under the conditions which necessarily prevail where the inexperienced wield the scalpel.

The hospital which does not assume some responsibility in the choice of medical attendants for those who come sick and helpless is shirking in its boasted cause of service and humanity. The hospital board which knowingly permits the incompetent and dishonest to treat and maltreat those who suffer, does an injustice both to the patient and to the cause of better surgery.

Dr. Lawrence W. Littig (Davenport, Iowa): In Iowa we have an immense number of small hospitals, with from twenty-five to fifty beds, and as a member of the Hospital Advisory Committee of the American Medical Association for Iowa, I have been looking into these small hospitals. Most of them have no resident physician; they do not care to have a resident physician. They have no pathological laboratory; they do not care to have a pathological laboratory. These hospitals are open to every physician or surgeon who wishes to bring a patient.

In one of these hospitals recently there was an operation for hernia by the chief of the staff. After the operation the operator asked me about the percentage of recurrences. I answered, "As you did that operation, 95 to 99. As the operation is ordinarily done, 2 or 3, or thereabout." He asked; "Why such a difference between my work and the operation as usually done?" I asked: "What operation did you do? Did you sew this to this, or did you simply include skin and subcutaneous fat?" He said: "My God, I just sewed skin and subcutaneous fat, didn't I?" "You did, and I watched every move you made."

What can be expected of a hospital where the chief of the staff does such a simple operation in such a manner?

Here is the thought I wish to leave with you. All of these hospitals have a training-school for nurses. If they have twenty beds, they have a training-school, and the course is three years. It occurs to me that the State Board of Health has a right to inspect these training-schools. A hospital cannot be very bad if the training-school be first class. We have a promise of a training-school investigation in Iowa; and I

believe an investigation will do an immense amount of good. The American Medical Association investigation is undertaken for the purpose of determining what hospitals are suitable for fifth-year work for medical students. As I said before, these small hospitals are not interested in fifth-year students and they cannot be reached through that channel; but they must have a training-school, and through the training-school much influence can be exerted, and the State Board of Health can exert that influence.

Dr. D. S. Fairchild (Clinton, Iowa): This question is absoabsolutely fundamental. It lies at the foundation of our expected or possible advancement in medicine and surgery.

It is not too ambitious for us to believe that some time America, instead of Europe, will be the center of medical education. The indications are that way, notwithstanding the misfortunes of the great war which came in Europe.

Dr. Littig has stated the facts as they really are, that there is no way of curing these abuses in the hospitals unless there is some authoritative body behind it. The mere fact of resolutions being passed amounts to absolutely nothing. The hospitals go on in the same way. The doors are all open to all sorts of enterprise; the hospitals are not equipped to do work in a correct way, and they do not care anything about it—it is simply a question of getting money, which is the fundamental factor in regard to many of these hospitals.

Gentlemen who practice in large cities and who have to deal with hospitals that have every facility, as far as laboratories are concerned, do not realize the difficulties that we have in some of our smaller cities in bringing about a better condition of affairs.

In my own particular case, for instance, I endeavored to bring about a correction of the conditions in our own state and our own community, and have made numerous attempts to bring the hospital people to a realization of the importance of doing these things. I appealed to the public press, and set forth in the newspapers what they were doing in the hospitals, with the result that every one of these hospitals in town invited me to stay away from their places, and now in our town there is not a hospital I can get into—I am an orphan! I am lost entirely, and simply because I undertook to bring this matter to the attention of the public; and I may say that the public entirely sympathized with me in this mat-

ter, but the hospital which is run for money alone is not in sympathy with such a movement.

This is simply an illustration of the conditions which exist in a great many of our communities.

The Board of Appraisers of the City of New York, as you know, had an investigation made of Bellevue and the allied hospitals, and they found that the percentage of accurate diagnoses as determined by the post-mortem examinations in cases where the patient died was only 47. This was on account of carelessness that crept into the institution; but, I imagine, it is so all over the country, and if we were today to take the diagnoses made in these hospitals and then subject them to the analysis of post-mortem examination we should find the same result, about 47 per cent of the diagnoses are correct and the others are wrong.

Now, this Association can pass resolutions, as many as they see fit, it will be helpful no doubt in bolstering up the men who are in power and authority; but, with the suggestions Dr. Littig has made here about putting this matter under the control of the State Board of Health, and excluding from their examinations nurses credited from these institutions, who are really maids-of-all work, instead of trained nurses, then we shall have achieved something that is definite.

The Journal-Lancet of Minnesota not long ago called attention to the fact that these nurses should be given a certificate as maids-of-all-work, instead of getting a certificate or diploma as graduates in nursing.

I am glad to hear this paper this afternoon because it calls our attention to a fundamental difficulty which concerns the American surgeon; and every one who has seen the work of good hospitals and good surgeons abroad sees how much better superintended are their hospitals than ours.

Dr. M. L. Harris (Chicago, Ill.): The conditions which have just been portrayed to us so vividly by Dr. Wetherill are really conditions not limited to any particular place or to any particular state or city, but are quite universal throughout the country. And I may say they are conditions for which the medical profession alone is responsible. The conditions will continue to exist until the medical profession assumes its full duty.

Hospitals during the past few years have sprung up in remarkable numbers all over the country. They have been organized and managed, and are managed many times, by

churches or religious orders,—I say this without any reflection on any religious order or any denomination,—but they are run, not for the benefit of the sick, but for the benefit of the organization that started them.

In some of our large cities this is so manifest that the different hospitals become simply bidding agents for the purpose of securing patients; and the men who are put on the staff at these hospitals—some of them, of course not all of them—are put on and are maintained there, not for the reputation which they bring to the hospital, not for the character of work which they do in the hospital, but for the number of patients that they can bring to the hospital that will pay the hospital fees.

It has been said by many that the medical profession should have nothing to do with the business management of the hospital, because doctors are not business men. If doctors are not business men it is high time they were learning to be business men.

After many years of experience, both in the management of hospitals and in being on the staff of hospitals, I am firmly convinced that no hospital can be properly run that is not run by and under the supervision and direction of business medical men.

I think every hospital should have on its board of directors or trustees or managers enough medical men to dominate in the policy and the management of that hospital.

As long as medical men will permit themselves to be dominated over by the lay trustees or directors of any hospital, the medical men are going to get the worst of it, and so are the patients.

No hospital can run without the doctor. I do not care who is managing it or under what denomination, the doctors have it in their power absolutely to dictate the policy of every single hospital in this country; and until they come forward and do it hospitals are not going to be properly run.

I am heartily in accord with this movement. I want to favor it and encourage it in every way possible. I think it is an exceedingly important matter, and I believe that we as individuals in our respective communities should urge it and take it upon ourselves to see that the management of the hospitals is dominated by medical men who are capable, in a business way and professionally, of doing so.

Dr. Carl E. Black (Jacksonville, Ill.): I want to say a word in favor of the small hospital. I work in a small hospital, one of fifty beds. I call your attention to the fact that most patients that receive hospital treatment in this country receive it in small hospitals, taken in the aggregate. Whatever we do we should keep the small hospital in mind. A few years ago we reorganized our hospital, and it was my privilege to write the by-laws for the new organization.

I thoroughly agree with Dr. Harris that the hospital should be run by the doctors. I think the time has come for the doctors to take over, in a larger degree, the business management of the hospital, and I believe there is plenty of business ability in the medical profession to do this.

In writing the by-laws of our hospital the staff were made the sole judges of its members, with the one limitation that only members of the county medical society, in good standing, could be elected to membership on the staff. It was provided that at least two members of the staff should be members of the executive committee. The executive committee consists of five doctors, two of whom must always be chosen from the staff. These provisions have not cured all the abuses and difficulties. In making every member of our county society eligible to membership on the staff we have done a great injustice to the surgery of the hospital because, unfortunately, all the members of the medical society are not surgeons, but a considerable majority of them do surgical operations just the same. In this matter we are greatly in need of help from such societies as the Western Surgical Association. You must help us solve this difficulty. How are we to separate those who are prepared to do surgery from those who are not prepared to do surgery? We have some men who go away and take a six weeks' post-graduate course, and are then ready to do anything, and others who do not even take the trouble to take a six weeks' post-graduate course.

We have no plan which places the surgery of the hospital in the hands of those who have sought to make themselves competent to take care of it.

In Illinois we have a limited inspection of the hospitals that have training-schools. The State Board of Registration of Nurses annually inspects all the hospitals that have training-schools in connection with them. This is more than the medical profession or the State Board of Health is doing for the hospitals.

Dr. J. N. Jackson (Kansas City, Mo.) : I believe Dr. Wetherill has presented the most important subject today that interests the medical profession, and particularly the surgical profession. We have for a good many years been concerning ourselves purely with the development of our scientific capacity, without realizing that we acquire capacity to accomplish work under conditions which are oftentimes absolutely hampering to a full development of scientific capacity.

We are coming now to where we are going to realize some of the requirements under which we work, and see whether the environment is such as to accomplish that work in the way we should.

No man would dispute anything Dr. Wetherill has said here today, that the real advance of true surgery to a higher plane is more hampered today by hospital conditions than by anything else.

I will make a statement with which I think no one will disagree, and that is, that it is always necessary for education to precede every reform and every step in legislation.

I am frank to say also that we cannot say after many years' experience with hospital work exactly what plan of organization is going to accomplish the reforms that we are working for, and probably it will take some time before that problem can be solved. But we have to have a campaign of education that is going to educate the people who are running the hospitals. We have been working for a good many years without a campaign of education, among ourselves, to control the education of our members; and we have accomplished a great deal, but I am frank to say to you that that which has accomplished more than all of our efforts for many, many years was that which was accomplished by the Carnegie Foundation.

For years and years we had our medical college associations, representing in many instances the best schools in this country; and each year, step by step, they made some little progress. But it was largely negative; it was not doing anything very much, and it was being done very slowly, but what happened when an outsider began to investigate the medical schools of this country and to publish the results of those observations? Within less than five years after the publication of that report we have found probably one-third of the inferior medical schools of America wiped out, one of the best steps that has been made in the progress in the history of medicine in America.

Now, I think the time has come when we want something of the same sort done relative to the hospital situation of America.

If we are going to improve the hospitals there are two things that are necessary. The first is to have a standardization of the surgeon by which we can recognize those men who are qualified to do the work in the hospital, and this is being attempted to be done for the first time through the College of Surgeons, which we hope will be successful.

But, after we get our standardization of what a surgeon is, it will accomplish nothing if hospitals pay no attention to this standardization—if the ignoramus, the untrained, the uneducated man has the same privilege, the same opportunity, that a man has who has had years of training. Under such conditions we cannot accomplish very much.

I do not believe there is a desire for an aristocracy, and, by degrees in the medical profession, to make a trust. I do not think there is anything in that. There is no opposition to any medical man, or to any man, doing surgery, providing that man simply is willing to spend the time to qualify himself.

There is no effort on the part of this Board to cut out the young man from doing work. We want to offer to the young man every opportunity to make himself a better man in surgery; and with the many schools and the many clinics and the many hospitals where a man who is ambitious can obtain an education in surgery, there is no excuse for a man to do major surgery who has not had preparation for it, except ignorance and slothfulness. Therefore not only the work of standardizing the surgeon is needed, but we should have the work of standardizing the hospitals, and I believe that any such movement must come through educational methods, that a resolution on our part endorsing this plan, to invite the Carnegie Foundation to investigate and standardize the hospitals of America, is a move in the right direction.

This does not have anything to do with trying to put big hospitals or little hospitals out of commission. It is not a question of the size of the hospital, though I am frank to concede that a hospital with ten beds may be run very much more efficiently possibly than a hospital with five hundred beds. It is not a question of size; it is a question of efficiency, and a move to increase the efficiency will be a move for the betterment of the small hospitals of the country towns, as much as it is in the large hospitals in our big communities.

President Davis: You will pardon me for making a few remarks. I consider this a tremendously big subject. It is one of those things that cannot be settled by a resolution or by the appointment of a committee or by discussion at a meeting of this kind, but it is one of the things that has got to come about gradually, and it must come about by a very thorough study of the situation from every standpoint. I thoroughly believe, as Dr. Jackson does, that the two things, the standardization of the hospital and the standardization of the surgeon, have got to go hand in hand; standardization of one will not mean much unless they can both be standardized.

It seems to me that we are getting to a time when, if we do not do something of this kind, our legislatures are going to open up and show up some of the abuses and do something in a drastic way, which is not going to benefit, but simply create a disturbance of all surgery and of the hospitals also.

It does seem to me that we ought in some way to get to work. I do not know exactly how it should be done, but we must get to work in such a way as to be ready to crystallize the thing in shape in order that some definite and positive agreement can be had among the hospitals of the country so that the different associations shall all be in harmony

I think the proposition to attempt to get the Carnegie Foundation to take this up is probably the best. Whether a simple resolution or the appointment of a committee would be best, we should especially recommend that they consider the thing, and perhaps should consult with the Foundation and see whether they cannot be gotten to work along this line.

But I do feel that it is immensely important, and I think we ought to do our best in trying to rectify the evil.

Dr. Wetherill (closing): I have given this matter much thought for many years. As I said before, as president of the State Society of Colorado in 1906, I referred to it as a local condition. Nothing whatever has been done locally, and under the circumstances I think perhaps nothing could have been done.

Whatever is accomplished is not going to be done in a day. It is an evolutionary process, and the benefit will come, not to this generation, in all probability, but to the next generation. It is therefore evident that most of the good will come

to the next generation, and most of the work that we have in mind must be done for the benefit of those of the future.

Many of you may know that a bill was introduced in the legislature of the State of Colorado about four years ago establishing a local standard for the practice of surgery. The bill was not passed. It was found to be impracticable, and was withdrawn; but it shows that the public is alive to the necessity for this sort of thing.

If we want to protect the interests of surgery and the interests of hospitals, let us do it ourselves, and not wait for somebody else to do it for us.

The American Medical Association, the American College of Surgeons, the Clinical College of Surgeons, and the Philadelphia County Medical Society have already put this matter up to the Carnegie Foundation, and I believe such a committee as has been suggested to work in co-operation with those bodies may ultimately accomplish something.

ANAPHYLACTIC REACTION FOLLOWING OPERATION FOR ECHINOCOCCIC CYST

Oliver J. Fay, M. D., F. A. C. S.

DES MOINES, IOWA

The idea of hydatid intoxication is not a new one; for it has long been accepted as a definite clinical entity, and was directly responsible for bringing exploratory puncture of supposed hydatid cysts and the so-called therapeutic aspiration into disrepute. But open operation for hydatid cysts, due care being taken to prevent the escape of the cyst contents, was specifically slated as innocuous by Achard[1] as early as 1888, and by Boudin[2] as late as 1910. Toxic incidents following open operations for hydatid cysts were not infrequently reported, but their significance was misinterpreted, due in part, perhaps, to the existing skepticism as regards the toxicity of the fluid. The troublesome, alarming, and even fatal phenomena observed following open operations were variously interpreted: they were attributed to nervous shock, to hepatic congestion, to decompression of the thoracic organs, to secondary infection, to peritonitic reaction, and to operative, anesthetic, or antiseptic shock. Not until 1902, when Lejars[3] reported toxic symptoms following a hydatid operation, was any reference made to post-operative hydatid intoxication; and not until another ten years had elapsed, and biologic discoveries had in part explained the mysteries of anaphylactic shock, did the idea of post-operative hydatid intoxication gain any surgical recognition.

Repeated experiments have demonstrated the absence or the extreme weakness of a direct toxicity in hydatid fluid, although occasional experiments, particularly when the fluid has been concentrated under vacuum to one-tenth of its volume, have resulted in the death of the guinea-pigs used when relatively very large doses have been employed. With Richet's[4] publication of the principles of anaphylaxis, a new field for hydatid research was opened, and it has been repeatedly demonstrated that under certain given conditions hydatid fluid will call forth a typical anaphylactic shock with a certain uniformity. While this anaphylactic reaction has been obtained under varying conditions, it seems to be most uniformly obtained when, as in experiments made by Boudin and Laroche,[5] the initial injection is made intraperitoneally, followed after the passage of some days by the intracerebral injection of a fractional dose. If the reaction is weak it may be increased by following the intracerebral injection, after the passage of a few moments, by an intraperitoneal one. This reaction is practically identical with the typical anaphylactic shock produced by animal and even by human experimentation.

Considering the very low degree of toxicity of the hydatid fluid, we may still hold, with the older writers, that the amount of fluid which would be absorbed during an open operation is insignificant, and is entirely insufficient to account for the grave and even fatal symptoms which may follow operation. Even with the most extreme care, however, it is impossible to prevent the absorption of the minimal dose of antigen required to call forth an anaphylactic shock, granted that our patient is sensitized and that the fluid has thus become an antigen.

We have already seen that under experimental conditions such a sensitization is possible. Does this ex-

perimental sensitization find a counterpart in the normal course of hydatid disease? We are accustomed to look upon the growth of an echinococcic cyst, except in the rare cases of hydatid phthisis, as a purely localized disease without systemic reaction, its symptoms of a mechanical order. In the late stages of hydatid disease the symptoms are those of a profound intoxication, though this condition may be accounted for in part by a functional defect of the liver or the digestive tract; and we have two proofs that hydatid disease, even in the earlier stages, is not without a constitutional effect. In addition to the presence of large numbers of eosinophiles in the tissues surrounding the cyst walls, marked eosinophilia is present in many cases of hydatid disease. (Chaffard et Vincent.[6]) Weinberg's complement fixation test,[7] while not absolutely to be relied upon as a means of diagnosis, gives sufficiently uniform results to warrant the belief that specific antibodies are present in very many, if not in all, cases. The toxins find their way through the cyst walls by dialysis, sensitizing the organism; and the absorption of a few drops of the fluid from a small fissure, during aspiration or open operation, is sufficient to call forth anaphylactic shock.

Dévé,[8] who collected 37 cases of post-operative hydatid intoxication, divides these cases into three groups: (1) benign, (2) grave, and (3) fatal, and the symptoms observed into the following eight groups:

1. Cutaneous symptoms, such as urticaria, various erythemas, edema and turgescence of the face, cyanosis, and profuse perspiration.

2. Symptoms of syncope, often associated with the third group.

3. Symptoms of collapse,—cyanosis, blotching,

cold extremities, ropy perspiration, impalpable pulse, and dilated pupils.

4. Gastro-intestinal symptoms, consisting in nausea, vomiting, anorexia, foul tongue, diarrhea, and tenesmus.

5. Abdominal symptoms, in which meteorism, abdominal facies, hepatic pain, and tenderness over the abdomen are prominent.

6. Respiratory symptoms, including dyspnea, anxiety, attacks of coughing, foamy expectoration, and pulmonary congestion.

7. Fever symptoms with their attendant chills, headache, and thirst.

8. Nervous manifestations, such as depression, prostration, agitation, coma, jactitation, or even epileptiform or tetaniform convulsions with trismus.

In cases of benign intoxication, fever and cutaneous symptoms, especially urticaria, are most characteristic. The fever usually rises suddenly, and may manifest itself at any time, from the first to the thirty-ninth day. The exanthema appears in the first twenty-four hours as a rule, though it may develop much later, and only occasionally persists for more than forty-eight hours. Respiratory difficulties,—dyspnea, temporary cyanosis, bronchitic phenomena,—are also common; and lesser nervous manifestations, consisting in agitation, momentary collapse, and abdominal pain, are also met with. The symptoms are usually transient, and while they may make the patient uncomfortable, they cause the physician no real anxiety.

In the grave cases, fever and cutaneous and respiratory symptoms are all of secondary importance; and the picture is dominated by symptoms of syncope and collapse. These symptoms are often accompanied, or even replaced, by nervous manifestations,—anxiety,

delirium, semicoma, and carphological movements. Respiratory symptoms sometimes become alarming; and occasional mention is made of gastro-intestinal disturbances. The symptoms usually develop soon after operation, and are of brief duration. They have been reported as late as the third day, however, and have sometimes persisted for a week.

In the fatal cases, fever, rising suddenly, is often the initial symptom; respiratory difficulties are prominent; and collapse is often present. Nervous symptoms, beginning with agitation and going on to delirium or loss of consciousness, are reported in many cases. Urticaria is absent. The symptoms usually set in immediately, or very soon after operation; and death ensues within twenty-four or forty-eight hours —in one case as late as the fifth day. Autopsy was performed in some of these cases, but no gross lesions were demonstrable; in some, the cyst cavity was found obliterated.

Had I had any knowledge of the symptom syndrome in post-operative hydatid intoxication, diagnosis would have been an easy matter in my own case, for its symptomatology was quite characteristic. The patient, a German farmer, 41 years of age, gave an entirely negative history, except for attacks of epigastric pain, severe but not paroxysmal, and accompanied by chills and fever. They extended over a period of nine years, and during the latter half of this period they had been accompanied by nausea, indigestion, abdominal tenderness, and jaundice, and had gradually increased in severity. A mass which had been palpated in the epigastrium at the time of the first attack had gradually increased in size. At operation the cyst, which contained about a liter and a half of fluid under considerable pressure, and hundreds of scolex-bearing daughter cysts, was found sit-

uated between the right and left lobes of the liver. Since it was impossible to remove the sac, it was swabbed out with pure phenol, followed by alcohol. It was then stitched to the peritoneum, packed and drained.

Two hours after operation the patient was fully conscious; his temperature was 101.8°; his pulse, 108; and respiration, 30. His skin was cold and dusky and he was bathed in perspiration. His condition was at first thought to be due to post-operative shock, but it remained unimproved throughout the next forty-eight hours, and frequent sedatives were required to control his extreme restlessness. His face was drawn and anxious, his extremities cold and dusky in spite of constant application of heat, and atropin failed to control the profuse perspiration. Great quantities of bile were draining from the cyst cavity, and there were absolutely no signs of peritonitis.

After the third day the patient gradually improved; but on the seventh day there was a return of the nervousness, cyanosis, and drenching sweats, and these symptoms persisted up to the fourteenth day. On the sixteenth day the patient was up and about, and on the twenty-fifth day returned to his home. At that time there was still a very free drainage of bile, but a month later only a sinus remained.

Experimental and clinical evidence has made it easy to accept an anaphylactic origin for these symptoms of post-operative intoxication; and, while the number of reported cases is thus far not large, representing, it would appear, only a small percentage of the actual number, the symptomatology has been sufficiently uniform to permit of the establishment of a definite syndrome and the making of a definite diagnosis. But here the thread ends, and the need for further research makes itself felt. Repeated doses of caffeine,

ether, or camphor oil, have been used, and artificial serum has also been employed; but none of these measures has proven adequate. General anesthesia has been suggested to control anaphylactic shock, and some experiments have seemed to indicate its effectiveness. Other writers affirm that it is useless; and at best its employment suffers certain limitations because of the element of danger when necessary to maintain deep anesthesia over a considerable period of time.

Extreme care in evacuating the cyst, walling it off, protecting the parietal wound, as well as the peritoneum, using a small, instead of a large, trocar—in short, the employment of every precautionary measure will, it is true, ward off any possible danger from direct intoxication; but the most painstaking care cannot prevent the absorption of sufficient fluid to call forth the anaphylactic reaction. When the danger of hydatid intoxication demanded that the exploratory puncture of cysts and aspiration therapy be abandoned, the surgeon looked to operation for the solution of the problem of hydatid treatment. Now that open operation, too, has been found to be fraught with danger, the problem is found to be a far more difficult one; for, though hydatid disease takes a slow course, it is, nevertheless, a fatal one.

Chauffard and Vincent state that the bearer of a hydatid cyst is "suspected" of being in an anaphylactic state when he gives a positive reaction to Weinberg's test; and it is possible that, with the further development and refinement of the complement-fixation test, we may hope to determine with some accuracy which patients are sensitized. But, then as now, I believe that we shall have to look to the working out of some pre-operative treatment which will desensitize the patient. Experimentally, the ground has already

been broken, and anaphylactic immunity been developed by a series of small injections, by a previous injection (subcutaneous) of serum heated to 100 degrees in a much diluted form, by the intraperitoneal injection of serum in a very small dose just preceding the injection of the second dose, and by an intrarectal injection of serum. A clinical method of developing anaphylactic immunity has yet to be realized.

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THE ADRENAL IN RELATION TO SHOCK

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When the adrenals from an animal not possessing accessory chromaffin substance are removed, the following syndrome soon presents: apathy, reduced sensibility, motor weakness, pallor, subnormal temperature, rapid, thready pulse, and irregular respiration. These words, transposed from Meltzer's description of traumatic shock, suggest the paramount importance of the suprarenal in overcoming that condition. Moreover, adrenalin is the vasoconstrictor for all blood-vessels, except those of the pulmonary circulation and possibly the brain. Seelig and Lyon have demonstrated vasoconstriction in the general circulation, and a shrinkage of all of the organs in traumatic shock, these reactions being comparable to that of adrenalin. We recognize pain, fright, emotional disturbances, and trauma as etiological factors in shock.

The experiments of Cannon and de la Paz demonstrate increased adrenalin in the blood as the result of emotional disturbances. Cannon explains the purpose of this by summing up the action of adrenalin,—i. e., adrenalin causes cessation of activities of the alimentary canal, and shifts the blood from the less insistent abdominal viscera to the organs immediately essential to life itself, such as the lungs, the heart, and the central nervous system. The increased cardiac vigor, the quick abolition of the effects of muscular fatigue, the mobilization of energy-giving sugar into

the circulation—these are the changes when fear or rage or pain causes the adrenal gland to pour its secretion into the blood. Crile attributes the increase of adrenalin content of the blood following fright to "phylogenetic association," and says "that fear born of innumerable injuries during the course of evolution has developed into portentious foreshadowing of possible injury and has become capable of arousing in the body all the defensive and offensive activities that favor the survival of the organism."

Cannon has shown an increase of adrenalin in the blood, not only from psychic disturbances, but as the result of nerve-stimulation, which is analogous to pain. In our own laboratory the presence of adrenalin in the blood with sciatic stimulation has been indicated in a common laboratory procedure. In this experiment, a very delicate oncometer was fitted to the kidney. On stimulation of the sciatic, the maximum blood-pressure was found to coincide with the minimum volume of the kidney.

Elliott (in the *Journal of Physiology,* vol. 44, page 374) teaches that the increased output of adrenalin with the exhaustion of the adrenal glands is the result of fright.

The clinical teaching of Crile has diffused the knowledge that psychic disturbances, the advent of foreign proteids in the blood, ether anesthesia, and operative trauma prolifically waste something necessary to maintain equilibrium of an individual under stress,—that is, that these things are the cause of shock. Crile has shown shock to be a terminal result, a permanent lesion not to be erased by normal salt, injection of stimulants, except possibly adrenalin, or by mechanical means.

Dreyer and Elliott have demonstrated that the discharge of adrenalin from the adrenals is reflex,—that

is, that afferent impulses must first reach some portion of the central nervous system finally to attain the adrenal by means of an efferent route. The efferent route they have demonstrated to be the splanchnic. The portion of the central nervous system where these impulses are transferred, is somewhere between the first thoracic nerve and the corpora quadrigemina.

Crile attributes a major rôle in shock to the brain-cell changes. While these occur, and undoubtedly have a peculiar significance, yet in the brains I have examined no absolute and complete destruction of cells sufficient, *per se,* to account for failure of adrenal function occurred. The Purkinje cells in some of the shocked animals had changes suggesting work (hyperchromatization), but never widespread exhaustion. The brain (from the work of Cannon and Elliott) is concerned, as Crile says, in driving the adrenals, but shock has been produced in animals with but little altered brain cells.

Elliott has shown that the adrenal content of normal glands varies more widely than the size of the medulla, and that the adrenal content of the normal gland fluctuates independently of its histology. He has also shown that fright diminishes the adrenal content. This author also shows that long-continued anesthesia diminishes the epinephrin (adrenalin) content of the gland; that damage to the brain leads to adrenal exhaustion; and that the toxins of diphtheria were capable of a like result. He has shown that exhaustion of the adrenal gland could be prevented by cutting the splanchnics above the diaphragm, and that direct stimulation of the splanchnic nerves was capable of increasing the adrenalin in the blood.

These facts led me to believe that the adrenal might be exhausted in shock. For this reason I set about to devise some satisfactory way of showing the changes

that occurred in the adrenal of animals subjected to various conditions. At first various histological methods were tried. Failing in this, we next turned to a study of the adrenalin content of glands from animals subjected to surgical procedure.

Elliott, in an article entitled "The Control of Suprarenal Glands by the Splanchnic Nerves," *(Journal of Physiology,* vol. 44), described a method of determining adrenalin content that is similar to the method that I had devised before I obtained knowledge of this article. This method, in brief, consists of the injection of just enough 1-100 extract of the adrenal gland to produce a rise of 10 mm. in the blood-pressure in dogs anesthetized with ether. The factor that we use is obtained by dividing the per kilo weight of the adrenal to be tested by the amount of gland that was necessary to raise one kilo of dog 10 mm. In deciding on this method the conclusions of Ried, Hunt, Beiberfield, Schultz, Levin, Sollman, and Brown were used as fundamentals.

By the physiologic method of determining the adrenalin content we found the guinea-pig and other animals with large adrenals to possess only a very small amount of active gland. This usually bore the same ratio to the body-weight as obtained in dogs. Further: a certain animal having unusually large adrenals had an epinephrin content exactly equal to that of other animals of the same species with smaller adrenals.

The results with surgical procedure obtained were as follows: The most striking exhaustion of the adrenal occurred in animals dying of typhoid infection, rabies, intestinal obstruction, and obstructive jaundice. To determine the rôle of anesthesia ether was administered for a period of four hours to six animals. These were not operated on, but were kept in warm,

padded boxes, and were evenly anesthetized. At the end of this period of time the adrenals were removed, and were found to have a greatly diminished epinephrin content, the average being equivalent to about 24 per cent of normal. In a series of animals where the peritoneum was traumatized, epinephrin exhaustion became extreme after four hours. The average content was 9 per cent of normal. These animals represent exhaustion, due both to trauma and to ether. The adrenals from all animals dying of primary anesthesia were examined. The average content in these was 6 per cent. This is assumed to be the cause of death, and the great adrenalin depletion to be due to exhaustion from fright. All of the animals dying in primary anesthesia were badly handled, frightened animals. Single primary operation with ether did not greatly reduce the epinephrin content, ether and operative work for four hours giving a figure of 21 per cent. However, a second operation with ether almost absolutely exhausted the gland. Whether the oligemia we find in shock is a separate factor, or whether this is a result of the spasmodic output of adrenalin that occurs in response to shock agents, is still open to discussion. It seems reasonable to assume that the repeated vasocontraction that occurs as the result of adrenalin output from repeated trauma, must have some deleterious effect on the vessel wall, and allow fluid and even blood to escape into the tissue. The protocols of the animal experiments are too voluminous to publish here; but in all of these epinephrin exhaustion was constant, and in all cases of traumatic shock either oligemia or increased specific gravity of blood occurred.

While this is a preliminary paper and is somewhat tentative, yet I think we are safe in assuming adrenal exhaustion to be a shock factor, and that epinephrin

is used to overcome the effects of shock. This assumption gives a ready explanation for the contributory shock rôle of ether, infection, fright, and pain.

DISCUSSION

Dr. James P. Aylen (Fargo, N. D.): In connection with this paper I only want to call attention to some observations that seem to bear out Dr. Corbett's general conclusion, that is, in connection with typhoid perforation, in operations at the time of perforation.

In my earlier practice I was struck by the fact that in most of these cases we had a slump for a few hours after the operation performed under ether anesthesia. This slump everyone has witnessed who has operated on these cases.

It was attributed, not so much to the operation, which the patients, considering their primary condition, stand very well, but to the slump that takes place within a few hours after operation and is quite pronounced. This was due, in my mind, not knowing of Dr. Corbett's conclusions or investigations, to the stimulation and the consequent reaction from the ether anesthia.

The anesthetic was changed, and chloroform was administered in these cases with some improvement in the condition, but it was not until, with a view of reducing the quantity of chloroform anesthesia, Dr. Crile's method of novocain and adrenalin was used that we got very pronounced improvement; and I might add that in these cases, while we attributed it solely to the relief of shock by limiting the anesthetic plus the "blocking," we also carried out the administration of adrenalin for some hours after operation.

These cases, we know, are more or less desperate, and I cannot say that our ultimate results were any better for the change of treatment, but we did not have the slump.

I think the profession will agree with me that the prognosis in operations for typhoid perforation depends pretty much upon the period that has elapsed between the time of perforation and the time of operation, but I am satisfied that the slump has been practically eliminated since ether was abandoned and chloroform anesthesia, fortified by adrenalin and novocain, was substituted.

This may have some bearing on Dr. Corbett's investigation.

Dr. A. E. Hertzler (Kansas City, Mo.): To discuss so scientific a paper is not a very pleasant job to have wished

onto one, but perhaps I can open this discussion with a part of my paper which is to follow.

I have been working along somewhat similar lines, in an empirical way, having in mind to find out some way to block the sympathetic tracts. Of course we know nothing about the exact path followed in transmission of painful impulses from the abdominal organs. Inasmuch as medullated fibres have never been demonstrated we may assume that the harmful impulses travel by way of the sympathetics.

I have tried chiefly with the idea of blocking the rami communicantes, thus blocking the trunk tracts before they reach the spinal cord. Experimentally perhaps this might have certain possibilities, but clinically it is impossible to do so with certainty.

Notwithstanding much has recently been written on the organic basis of shock, I believe I am safe in saying that up to date none has been established. Like changes are found in certain intoxications, in fatigue or even following ordinary functional activity stopping far short of fatigue. The recognition of these changes, though but recently brought to the attention of the medical profession, is not new. Not everyone is familiar with the fact that the best work on cell changes on the brain dates back some twenty or twenty-five years ago. The work done at that time is considerably more complete, and, I think, more exacting and more scientific, than the more recent and better known work of Dolly, upon which the work of Crile is largely based.

So I believe, taking these things into consideration, we have to turn for our final hope to the chemistry of the various physiological functions in shock and allied conditions. For this reason I believe Dr. Corbett's paper is very well worthy of attention.

It is exceedingly interesting to note from Dr. Corbett's experiments the astonishing effect of ether alone. As that is exactly in accord with the drift of my paper, I am therefore, of course, only too glad to endorse it. The paper, of course, is, as all scientific works should be, prepared without any immediate intention of straining it to any clinical application. Yet it agrees with what we have long known regarding the action of ether, judging from clinical observation. The paper therefore becomes at once of practical importance.

Medicine suffers too much from the mental attitude of the investigator, in which he has in mind a certain clinical possibility; and his scientific results are too apt to bear the color-

ing of the hope which he expects to realize. Dr. Corbett is therefore to be commended for his modesty in his estimation of the importance of his investigations.

The results of the stimulation of the operation in preventing the immediate adrenal absorption under the effects of ether are exceedingly interesting, and may possibly explain some of those cases of death in which the patient recovers from the anesthetic, but dies, after a period of hours, without there being any demonstrable pathologic lesion. Death may be due to adrenal exhaustion. I have had this happen not less than three times in simple cervix and perineal repair, in patients that gave no particular difficulty at the time of operation on account of the anesthetic. This is quite sufficient, I think, to explain why I recently elected to do these operations all under local anesthesia.

Dr. A. W. Abbott (Minneapolis, Minn.): I would like to ask Dr. Corbett to say, when he closes the discussion, whether he made experiments relative to the time of recovery from the loss of adrenalin.

Dr. Corbett (closing): The purpose of this work was to determine the power of certain surgical procedures, to reduce the epinephrin content of the adrenal, and to determine the importance of adrenal exhaustion in shock.

The exhaustion of the adrenal following the prolonged administration of ether is very marked. In comparison to this, the immediate exhaustion following peritoneal trauma is surprisingly slight. However, this becomes accentuated in the hours following operation. Twenty-four hours after operation the content of the adrenal seems to be at a very low ebb.

Infection, and particularly typhoid infection, reduces the epinephrin content. Therefore, one might hesitate to use ether in operating on typhoid. Further than this, the danger of ether anesthesia for operation following primary operation after an interval of twenty-four hours, is emphasized.

As to how the adrenals are exhausted, very little has been definitely proven. Elliott has demonstrated that cutting the splanchnics above the diaphragm prevents the exhaustion of the adrenal. In addition, it has been proven that a certain portion of the central nervous system is necessary to bring on this exhaustion of the adrenal gland with ether or infection—that is, we cannot exhaust the adrenal after cutting the cord any distance below the corpora quadrigemina. The removal of the brain above this does not seem to interfere

with the adrenal exhaustion. Morphine prevents shock and prevents adrenal exhaustion. To emphasize this, I took Crile's chart where he shows the result of continuous traumatization of the peritoneum of animals under ether for several hours. In comparison with this, I compiled the data of a series of animals anesthetized with ether and morphine, and traumatized for a like period of time. I found hour by hour that the average blood-pressure with ether and morphine was much higher than with ether alone, and that the figures compared almost exactly with those given by Crile for nitrous oxide.

As to the time it takes the glands to recover, I have no data whatever. Chloroform seems to exhaust the adrenals in about the same proportion as ether. A study of nitrous oxide anesthesia is now under way.

LOCAL ANESTHESIA IN THE PREVENTION OF AFTER-PAIN AND SHOCK

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About all I expect to do in my remarks is to give an outline so that Dr. Harris, who is to follow, may have a suitable text and pretext. By drawing forth his fund of wisdom I hope to serve the cause of local anesthesia in the control of after-pain and shock.

I may illustrate the duration of the pain by diagram on the blackboard. I will make the study over a ten-hour period, simply an arbitrary calculation.

Say this line (drawing) represents the first two hours. We give one-sixth of a grain of morphine before the operation begins, as is ordinarily done in major operations under local anesthesia. You operate under novocain, and at the end of the two hours, two-tenths of the time has elapsed. At the end of this time the patient has still full effect of the local action of the novocain plus the action of the one-sixth of morphine, so he is perfectly happy and altogether comfortable.

Soon after this time the novocain begins to wear off, so at the end of four hours,—that is, after four-tenths of the time has elapsed,—the novocain is entirely worn off, and he has six-tenths of the time to go on with his one-sixth of a grain of morphine. The degree of pain experienced will then depend on the sensitiveness of the individual and the character of the operation.

Ordinarily speaking, then, at the termination of the six-tenths of the time the patient is suffering from the full effects of the type of the operation which has been performed, save as modified by the morphine. In other words, the anesthetic no longer plays any part in the control of the after-pain.

Now, when the patient is operated on under ether, at the end of two hours the patient is awake and is suffering from the full effects of the operation. Likewise he is waking up from his drunk from the ether, and not only does he experience the full effect of the pain resulting from the operation, but he has lost his orientation, and he is profuse in his complaints of pain. Compared with the novocain patient, the relative discomfort of the patient operated on under ether is very striking.

In order to have a fair parallel to judge by, we should also give the ether patient morphine before the anesthesia is begun. When he awakens he will have his pain modified by the morphine.

Now, we have at the end of this lapse of time the patient recovered from his anesthetic as far as the sleep goes, and he is wearing off gradually his morphine, so that, when we come to the end of this period of ten-tenths of the time, the anesthetic in both patients is worn off, and the effect of the morphine, generally speaking, is likewise worn off.

So that we have, then, so far as the relative comfort of the patient so far as after-pain from the wound is concerned, no difference between the one operated on under local anesthesia and the one operated on under ether. But the one patient still is sick from the ether; and to the degree he is still bothered with the ether he is more uncomfortable on that account than the patient operated on under local anesthesia.

And it is this factor here (nausea), in my opinion,

that furnishes the main difference as regards after-discomfort between the two types of anesthesia; in other words, in doing the operation under local anesthesia with novocain the patient escapes the disagreeable after-effects of the ether.

The only method of effectually controlling after-pain by the use of local anesthesia is by the use of quinine hydrochlorid. With that drug we are able to control the after-pain for varying periods of time. But quinine is the crankiest drug which we have to deal with in local anesthesia.

Experimentally, I can produce local anesthesia varying anywhere from two hours to two weeks by varying the amount of drug and its combination or not with adrenalin. Under conditions of the operating-room such definite results cannot be secured. Nevertheless, much can be done to control the after-pain, once the operator is familiar with the use of this drug.

Those who employ local anesthesia simultaneously with a general anesthetic have no means of knowing what effect the local anesthetic has, if any, on the comfort and well-being of the patient. General impressions and "statistics" produced under those conditions are subject to the very wide influence of the "personal equation."

It is claimed, for instance, by those that advocate combined anesthesia that quinine is effective in one-sixth per cent solution, even in loose connective tissue. This drug in this strength is not effective in operating upon patients when not under the influence of a general anesthetic. Does the general anesthetic have some peculiar influence that augments the action of the quinine?

The only way that one could get an effect from local anesthesia sufficiently prolonged to have any influence in the control of after-pain is to get it directly

in contact with nerve-bearing tissue. It is useless to inject the loose tissue, which has few or no nerves.

The use of local anesthetics in the control of after-pain may be illustrated by citing a specific operation. In the operation for hemorrhoids, for instance, the injection must be made in the exact site of ligation. If the nerves are blocked centralward from the point of ligation the effect is more fleeting. The site of ligation may lie in the field of hyperesthesia, even, if central blocking is done, thus augmenting rather than limiting the after-pain. This does not apply when the nerve trunk is actually injected.

In order to secure a lasting anesthesia with quinine one must have at least a half-per-cent solution. In order to be certain of a lasting effect a one-per-cent solution must be used. Solutions of a strength less than one per cent, if skillfully used may be effective during the operation, but the effect is not lasting. Even for this less than one-half of one per cent is uncertain, and less than a fourth of one per cent is totally unreliable under any condition.

If injected directly into the nerve-bearing area, as in the papillary layer of the skin or into a nerve trunk the anesthesia is likely to be more permanent. Yet, after all, one cannot exactly calculate the time the anesthesia will last; but, outside of that, I believe that the use of quinine in prevention of after-pain is a poorly supported hypothesis. In weak solutions, ⅙ per cent or less, there is no satisfactory proof that quinine produces an effective anesthesia, no matter how injected or in what tissue, let it be repeated.

That is particularly true when injected into loose tissue, where quinine produces enormous exudation of granular fibre, likely to cause all sorts of mechanical disturbance. I would like to suggest to anyone who uses quinine in the peritoneum that they try first on animals and see how the drug really works.

Now, say here on the diagram is the abdominal wall united, and here is the peritoneum. The directions given by some operators is to put the quinine in the inverted peritoneum. In order to effectually anesthetize this area one must get in above the transversalis muscle. The nerves do not come along immediately below the peritoneum, but they come down above the transverse muscle. When an attempt is made to block them according to the directions given, the results must be very uncertain.

What happens if you do inject quinine sub-peritoneally if that point is a fibrous exudate, which in animals will pull loose the peritoneal suture, and a gaping peritoneum at this point results?

So far as controlling the after-pain in abdominal operations goes, we are obliged to block off the nerves, at the site of operation, or at the rami communicantes. In the human subject this is difficult to do with certainty. The anatomic relation of the ramus communicans is definite to be sure, but if one injects for an abdominal operation one must inject from 4 to 6 places on each side. This takes a lot of time, and is trying both on the part of the operator and on the part of the patient; and before one gets that done one will probably have produced more injury than with a general anesthetic.

In order to control after-pain, of course the simple thing would be to block these areas within the abdomen, which could be done only in relatively simple cases. But when one gets in the real shock-producing area down in the pelvis in the region of the gall-bladder or when there are adherent tumors or inflammatory masses in the pelvis, one cannot get within reach of these nerves in order to inject them. It is in those cases, therefore, where we have learned to expect shock that we are powerless to block off the afferent

nerves. Therefore we have to confine ourselves to deft operating under ether until we find more effective ways for managing these cases.

It is often stated that with local anesthesia we have no shock. This is true to a certain degree. While there is no dangerous degree of shock, yet sometimes our patients flatten out. I have never seen a patient in any serious condition; yet, after all, there is that depression which reminds one of shock. This is true in operations in which the patient complains of no pain. I believe fully nine-tenths of the profession still believe that when one speaks of operating without any pain under local anesthesia he is a candidate for the Ananias Club. Some operations of magnitude, particularly about the neck, for instance, as in blocking out the neck for carcinoma of the jaw, combined with resection of the jaw can be done under local anesthesia without any pain at all; yet some of these patients flatten out, and they are mighty glad when you are through, although persisting in saying that they suffer no pain.

In certain types of goiter, too, particularly in those in which some person has ligated the superior thyroid vessel, or has started in with ether, and cut down upon the gland, in other words, where scar tissue makes the operation tedious, the patient is apt to show the effect of the operation.

In carcinoma of the rectum, too, where the carcinoma is relatively high up, although one causes the patient no pain, he begins to flatten out even before one begins to make a traction.

On the other hand, one can do any sort of perineal operation or operation on the cervix or uterus, the Freund-Wertheim, or operations of like magnitude, and the patient is perfectly fine afterwards, and this flattening out after-effect is absent. The same thing

is true in hernias. In umbilical hernias, for instance, requiring a long incision, with vast and extensive adhesions, with an opening as big as a saucer, the patient stands all the necessary manipulation without any complaint and without flattening out.

So the question arises, what does this? Sometimes the novocain injection is followed by temporary disturbance, for instance, in enucleation of the tonsils. I have noted repeatedly, in preparing for tonsillectomy, that these patients often have much the same symptoms that these flattening patients had. The novocain is injected in the loose tissue,—which readily absorbs the drug, and the patient gets the full effects of the novocain.

I have, therefore, made attempts to limit the use of novocain. Except in very rare instances, I limit the amount used to five grains. I figure out before beginning the operation where I can best use this amount of novocain and do the rest of the operation with quinine. For instance, in umbilical hernia, here the delicate part of the operation is where the adhesions are. The long incision across the belly, no matter what the extent, is infiltrated with quinine. The novocain is used in blocking off the ring of the hernia sac. I believe in this way, by limiting the amount of novocain used, we are able to prevent the pain and post-operative flattening out of the patient.

There is one other point not exactly belonging to my subject. I bring this up in order to make the way clear for Dr. Harris, if he chooses to use it as a basis for his remarks. That is the combination of local and general anesthesia. There are very few operations in which I plan to use the combination. Now, in carcinomas of the rectum, for instance, if they are high, that is, if they are higher than I can reach with my finger, I begin with the local anesthesia with the deliberate

intention of finishing the operation with ether. The carcinoma is loosened from below, and then the last stages of the operation are done under ether, either through the perineum or through the abdomen. However, to start in with an operation and go as far as one can with the idea of finishing the operation under a general anesthetic I believe is a bad plan as a general principle. The patient may be excited if pain has been produced and the anesthetist is apt to hasten the anesthesia. This makes a combination of factors very apt to spell trouble.

The question of local anesthesia to prevent after-pain when the patient is operated on under a general anesthetic has been covered in a way with what I have said previously. The same principles apply. Some six or eight years ago one of my associates at that time, Dr. Rogers, suggested that it would be a good stunt to use quinine deliberately to prevent after-pain; and we began a series of experiments designed to control the after-pain in patients upon whom it is ordinarily necessary to employ a general anesthetic, and the principles which we outlined are as I have stated here.

You have to go at it with a definite purpose and use the quinine just right, in sufficient strength to get any actual results.

The prevention of gas pains is often credited to local anesthesia. It is often stated that patients operated on under local anesthesia have no gas pains whatever. I wish this were true, but it is not. Inasmuch as only the simpler abdominal operations are done under local anesthesia the test is an unfair one. Yet in some of the simpler operations the most troublesome and annoying gas pains have followed absolutely painless operations. This is true even of hernia operations.

I recently operated upon a young doctor for hernia

under local anesthesia. He said he felt no pain whatever during the operation; but he did have very annoying gas pains for a day or two.

So far as using novocain in the skin to control after-pains, when the patient has been operated on under general anesthesia, it is not worth the time that one wastes in using it. It lasted at most two hours, at least in the weak solutions generally employed and the general anesthetic usually covers that period.

The same thing is true, I am satisfied, with the one-sixth of one per cent of quinine sometimes recommended. At least I have used one-sixth per cent time and again in my own leg and the anesthesia is incomplete and fleeting. It lasted, at most, not more than a couple of hours. Until those who claim that it does last longer will operate under it alone and determine the exact duration of its action, I believe I am justified in being skeptical regarding the accuracy of these observations.

DISCUSSION

Dr. M. L. Harris (Chicago, Ill.): The subject of local anesthesia or nerve-blocking is one in which I have been interested of late. I have done three hundred and fifty major operations under nerve-blocking, and a large number of minor operations, of which I have not kept track.

The question of after-pain following operations done under nerve-blocking or local anesthesia I shall consider first; and I should like to make a distinction, if I may, between local anesthesia and nerve-blocking.

Local anesthesia is an infiltration anesthesia; nerve-blocking has nothing to do with the region in which we operate, but refers to the blocking of the nerve at some point between the field of operation and the spinal cord. It may be quite remote from the field,—for instance, when we operate on the foot by anesthetizing the sciatica high up in the thigh.

After-pain, in my experience, is purely individual. One patient after an operation done with nerve-blocking will complain of after-pain, and another patient with identically the same operation, blocked in identically the same way, will claim

that he has no pain whatever at the seat of the operation; and this, whether the operation has been done by infiltration or by nerve-blocking.

I explain this in the same way that we explain the difference in the degree of complaint made by different patients in regard to pain. We know that one person will complain excessively, while another patient with practically the same condition will make no complaint at all. In other words, the amount of after-pain depends upon the sensibility of the central-nerve organs of the particular patient.

The after-pain in an operation is simply the result of the irritation of the local trauma. If you make an incision in your skin you feel a burning pain. After an operation under nerve-blocking, when the effect of the blocking has entirely disappeared, the patient will complain of the same burning sensation that he would have complained of had he been cut without any nerve-blocking. And, as I say, the degree of complaint or the amount of pain which he will have will depend entirely upon the sensitiveness of his central nervous system. If he is obtuse to ordinary sense-impressions he will complain of almost no pain. If it is a highly organized and highly sensitive woman,—or man either, for that matter,—the patient will complain considerably of the pain at the seat of the operation.

That, too, is demonstrated again by the fact that if we give these patients something which benumbs their central sense organs,—morphine or veronal, or any similar substance,—we find just to the extent to which their sensory organs are benumbed will they complain less of the local sense of pain.

For a time I injected quinine and urea at the seat of the operation after I had finished the operation. I tried it particularly in hemorrhoids, as has been mentioned; and it gave me so little satisfaction that I discontinued it. I never operate on hemorrhoids now, or anything about the anus or rectum, by local anesthesia, but always by nerve-blocking. I always block the sacral plexus, which is a very simple thing to do, and one of the most satisfactory things, I think, in the whole field of nerve-blocking. In that way we have no edema, no infiltration, or anything about the field of operation.

After a few hours a patient who has been operated on for hemorrhoids will gradually begin to feel pain. The length of time in which the pain comes on after an operation of that kind will depend entirely on the character of the fluid with

which you have blocked the plexus,—in other words, how long your blocking lasts.

We can make the blocking last from a few minutes to several hours, but after the effect of blocking begins to wear off, the patient will have more or less pain at the seat of the operation.

So I do here just as we do after general anesthesia, introduce a suppository after the operation; and these patients get along without any pain, of course, due to the benumbing effect of the opium or what you put in your suppository.

So, then, I think that the degree of after-pain, other things being equal, depends on the susceptibility of the central nervous organization of that particular patient. Gas pains, which the Doctor mentioned, are not prevented by nerve-blocking or local anesthesia. That has been my experience. In a laparotomy for any purpose, done under nerve-blocking or local anesthesia, one will have gas pains perhaps as frequently as after the same operation done under general anesthesia.

I do not think the pains are so severe, or amount to anywhere near so much, as when done under ether, where we have ether eliminated through the intestinal tract as well; but these patients do have gas pains, so that the gas pains must be due to the effect of our manipulation of the abdominal contents.

As to the question of shock following nerve-blocking. The question of shock is a most interesting one, and a most difficult one for us to figure out, as we have learned here today from the very excellent paper by Dr. Corbett. I want to warn, however, against the tendency to generalize on the subject of shock. I do not think that the Doctor did, and I do not want to be misunderstood in this connection, but there was a tendency for his paper to leave the impression that the amount of epinephrin in the adrenals was in a sense synonymous with shock. Now, it is not anything of the kind, nor did the Doctor say so. It is possibly one factor in shock, but only one; and there are a great many more; nor is it proven that the mere percentage of epinephrin is related directly to shock. It may be the adrenals become exhausted in shock, but it is not shown that the mere epinephrin content, whether greater or less, is synonymous with shock.

Without going into the theoretical part of it, in practice shock is due to two conditions,—afferent impulses and toxic agents. Perhaps you may be surprised to know that I have

left hemorrhage out of the question of shock. I exclude hemorrhage from the production of shock because I call a condition due to loss of blood not shock but collapse. That may appear a distinction without any difference, but I do not think so; and unless I am digressing too far from the subject I should like to explain what I mean.

Collapse is a condition, we will say, of depression of all of the vital powers. We say shock is the same; and, so far as definition goes, they are the same, but there is this very great difference: In collapse the forces may be restored. For instance, collapse due to simple hemorrhage is recovered from when the loss of blood is restored, while in pure shock the forces cannot be restored except after the expiration of time, showing that there is an exhaustion of something in the system which it takes time to reproduce; and that, in my opinion, is the great distinction between collapse and real shock. A patient comes into a hospital in shock from a crushing injury, or we may eliminate the crushing injury because there is always more or less hemorrhage, say in a patient in shock from a burn, a superficial burn, over an area sufficiently large to precipitate the patient into intense shock, and I know of no more perfect picture of shock than that following an extensive burn,—a shock due primarily and solely to afferent impulses. There is nothing that we can do to that patient that will restore him from that condition of shock except the element of time, although he may not have lost a drop of blood. No transfusion, or anything, will take that patient out of the shock. There is an exhaustion of something in that individual that gives him power and strength, and nothing but time will restore it.

Now, operations done under nerve-blocking, if we block perfectly,—I say *if,* because with all the experience I have had I know that I cannot block perfectly in all operations,—but if we block perfectly, we can operate in the area that is blocked without the production of shock.

If there is loss of blood, of course, there comes in the question of hemorrhage, and if that blood is not restored we have practically the condition of shock; but, eliminating entirely the question now of hemorrhage, if we block completely we can operate without shock.

In the 350 major operations which I have done under blocking, the pulse has actually undergone a decrease in the number of beats, with the maintenance of blood-pressure in 39 per cent of the cases, and has remained identically the same,

no change in the count in 13 per cent and a fraction, or in 53 per cent of the cases the pulse has undergone no change whatever nor any decrease. In 47 per cent there has been an increase varying from three to four beats up to thirty or forty, in some very severe operations. This, too, in abdominal cases. Just recently I removed the cecum, the ascending colon, the hepatic flexure, and part of the transverse colon for a large carcinoma of the cecum and first portion of the ascending colon, and made an ileocolostomy in a person sixty years of age. He went on the table with a pulse of seventy-two, and had a pulse of seventy-four when he reached his room after the operation. The operation took me an hour and a half, and was done by blocking the nerves at their exit from the intervertebral foramina. By blocking the ninth, tenth, eleventh, and twelfth dorsal and first lumbar nerves on the right side, we had a section of the body which was completely anesthetic, and that was all the anesthetic he had. He was perfectly conscious all the time, and talked about the case,—how we were getting along, etc.

Concerning the "flattening out" of the patients mentioned by Dr. Hertzler: Take any person and let him lie in a cramped position and keep perfectly still, or try to, while doing an operation of an hour or an hour and a half on him, and see how he will feel.

I allow my patients to move. If they want to straighten out their legs, or want to draw them up, I tell them to do it, so long as it does not interfere with the operation. Let them lie in the most comfortable position possible. If the patient wants a drink during the operation and there is no contraindication, I allow him to have it. Make the patient as comfortable as you can while the operation is going on, and he will come off the table without so much "flattening out."

It should be remembered that all of the substances which we use to produce nerve-blocking are toxic. If we could discover a substance which is not toxic that would block the nerves, we could block practically any area in the body. But we have no non-toxic substance, and when we use considerable quantities of novocain we shall get in some patients the toxic effect of the drug, but so far I have never seen anything that approached a serious condition from the toxic effects of the novocain, and I have used as much as a gram of novocain in a single operation.

I will say that that amount of novocain is a large amount, and if injected alone might produce serious toxic effect in

perhaps any individual. But it has been found by experiment that the toxic effect of novocain is due largely to the rapidity of its absorption. An amount of novocain that, if absorbed quickly, will produce serious toxic effects, will, if absorbed slowly, produce practically none. As a corollary to that, the toxic effect will also vary according to the absorptive powers of the region in which you inject. In some regions novocain is absorbed much more rapidly than it is in others. I use a solution of novocain which is absorbed very slowly. I have two basic solutions of novocain, one a half of one per cent and the other a one per cent. Both of course are made with distilled water.

It may be elementary, but this is so important and there is such a tendency to take things for granted, and then get bad results by not following the most minute details, if you will pardon me I will say that water is the most difficult thing there is that we have to sterilize. Most serious results, such as sloughing, which occasionally follows the use of this solution, or of normal salt solution for that matter, as ordinarily prepared, is due to the fact that the water with which it was prepared was not sterilized. That has been demonstrated over and over again. The simple bringing of water to the boiling-point does not sterilize it.

Distilled water is fractionally sterilized, or else it is sterilized for at least thirty minutes in a high-pressure sterilizer. That is all done before the novocain is put in; and after the novocain is put in it is never boiled for longer than three minutes. It is said that novocain will stand boiling. It will stand boiling for a short time, but if you boil it a long time the novocain loses its effect, and the longer you boil it the less its effect.

After these solutions have been sterilized in this way and allowed to cool to 160° or below, one per cent of chlorbutanol is added. This is more than will dissolve, because this substance is soluble in only eight parts per thousand. A simple way of insuring a saturated solution is to add one per cent; if some settles in the bottom as crystals it is perfectly harmless. Then I have two other basic solutions, one a two per cent solution of calcium chloride in distilled water, and a two per cent solution of magnesium chloride. These are made separately, so they may be mixed in varying proportions as may be desired for the particular case.

For all ordinary operations, the half of one per cent novocain solution is sufficient, and to three parts of this half per

cent solution I add one part of the calcium chloride solution, and a half of one part of magnesium chloride solution. This reduces our novocain solution, as you see, to less than three-eighths of one per cent, or to a little better than a quarter of one per cent solution.

Calcium chloride I have found greatly intensifies and prolongs the effect of the novocain. It delays the absorption of the fluid, so that it remains longer in contact with the nerve, and, second, we find that calcium and magnesium are the salts of the body which act as the damper on cell- or nerve-irritability. As the calcium content goes down nerve-irritability comes up; as the calcium content goes up nerve-irritability goes down. That is what led me to use the calcium chloride. With the calcium chloride you can produce an anesthesia that will last four hours; while the same amount of novocain put in alone will not last over twenty or thirty minutes.

I have found that the magnesium increased the rapidity of the action of the novocain. For instance, a novocain and calcium chloride solution that will take, as it will in some of the large nerve-trunks, thirty minutes to produce a perfect blocking, if we add the magnesium chloride the same amount of novocain will produce a blocking in ten minutes, showing the effect of the magnesium in increasing the rapidity of the action of the novocain.

When we want to block a large nerve-trunk like the sciatic or the anterior crural, or want to block the brachial plexus or the sacral plexus, I use the one per cent solution of novocain as the basis. This diluted in the same way gives up a little less than a five-eighths per cent solution of novocain. That will block the sacral plexus. If you use enough of the fluid you can block the entire sciatic nerve, taking all of the sacral plexus, so that the anesthesia will reach to the bottom of the feet.

I am asked where I inject the sacral plexus. The solution is injected into the sacral canal at a point between the sacrum and coccyx.

If we catch the third sacral we will have anesthesia reaching to the top of the symphysis; and we will have an anesthesia that will reach down to the heels if we catch the second sacral; while, if we get up as high as the first sacral, we will have the entire distribution of the sciatic anesthetized.

As I said, in a blocking that is complete there is no shock. I have blocked the anterior crural at Poupart's and the sciatic,

high up, and made thigh amputations. I have done four thigh amputations in old people, with gangrene of the extremity without their feeling a particle of pain or knowing practically that the operation was done, and without any shock. We know that in these old people with gangrene, hardened arteries, diseased kidneys, etc., a general anesthetic is apt to kill them; but I have done four of these operations with nerve-blocking without any shock and without any apparent effect on the patients.

I have not used spinal anesthesia, and the chief reason is that we have no way of limiting the effects of the drug when put into the spinal canal. Spinal anesthesia is a central anesthesia, and not a peripheral one. What we want in nerve-blocking is peripheral anesthesia; in other words, when we anesthetize in the spinal canal we are anesthetizing nerve-centers, and not blocking nerve-trunks. In the central spinal canal the fluid circulates.

Anything that is put in the central spinal canal will reach the ventricles of the brain in a short time, it does not make any difference whether the substance is insoluble or soluble.

If we could limit the effects of the anesthetic to the point we inject, I think spinal anesthesia would be ideal. But, until we discover some way of doing that, I look upon spinal anesthesia as very dangerous, and of course the statistics show that it is dangerous.

In abdominal operations, all of the abdominal area and the pelvic area at the present time can not be blocked, at least I am not able to do it; but, I think, it is due entirely to ignorance and lack of experience and skill. I think some day we shall be able to block these areas the same as we are able to block other areas.

The tracts by which the spinal nerves reach the abdominal viscera are not fully known.

By blocking the seventh, eighth, and ninth dorsal, and perhaps the sixth, we will have the region of the gall-bladder blocked, and that region may be operated on without any pain.

The most difficult part to block is the pelvic region, the lateral walls of the pelvis and the posterior wall of the abdomen. We know very well now what areas in the abdominal cavity are sensitive and what are not, and we can do a great deal in the abdomen by care in avoiding those regions which are painful.

There is no trouble in blocking the region of the appendix and the cecum.

In abdominal operations that involve regions that we know to be sensitive, or know are not blocked, I give the patient a little gas and oxygen during that part of the operation which is painful. As soon as the gas is stopped the patient wakes right up, and you go ahead with the operation, do the sewing up, etc., without any pain to the patient.

The bladder can be blocked without difficulty. If you are going to do a suprapubic operation, and there is nothing the matter with the base of the bladder, simply block above. If you are going to do a prostatectomy, block the sacral plexus.

The reason that there is some shock connected with operations under blocking is due to hemorrhage which is not affected in any way by the blocking, and to the toxic effect of the substance used and to the fact that we work at times in regions and areas which are not blocked.

I am asked if I add adrenalin. I forgot to mention that I add adrenalin to the solution just before it is used. The amount I use is five minims of the one to one-thousand solution to thirty c.c. of fluid injected.

I am asked if I expose the nerve tracts before injection. No, I do not expose them

Dr. A. A. Law (Minneapolis, Minn.): The University of Minnesota Clinic has followed with interest the work of Dr. Crile, Dr. Harris, Dr. Hertzler, and the various surgeons abroad who are exploiting local anesthesia. We have followed, as well, the various methods of exhibiting them,—the nerve-blocking, infiltration, Bier's venous anesthesia, etc. We have repeatedly used every method with the single exception of intraspinal anesthesia, and we have had no inclination to use this latter. We have done many of the major operations under local anesthesia; and therefore we do not in the least deprecate its use, but, on the contrary, enthusiastically advocate it. Recently, however, we have had our attention called to some unhappy experiences.

I have had none personally, but I am cognizant of one death following the injection of half of one per cent novocain into the perirectal region, preceding an operation for hemorrhoids. Unquestionably, the drug was injected into the hemorrhoidal veins, and was picked up immediately by the circulation. I know of one case of death and two cases of alarming toxemia following the use of novocain in the opera-

tion for the enucleation of tonsils. I had an example of the toxic effects of novocain in my own family ten days ago, which nearly resulted fatally.

A dentist's office telephoned me at the University Hospital that while this patient was in the dentist's chair they had injected novocain into the alveolar process, and that she had gone into collapse, and asked what they were to do.

Dr. Moore and I drove across Minneapolis at top speed, and found this patient in utter collapse. Her color was dusky and her respiration was labored, but her heart was good. She had complete motor and sensory paralysis from the navel down, which persisted for an hour. She had been unconscious before we arrived, and had exhibited all of the symptoms of respiratory paralysis, viz.: labored breathing, cyanosis, and all of the auxiliary muscles of respiration called into play.

Since these experiments Dr. Corbett and myself have been sounding a note of warning to our students, not deprecating in the least the use of this agent and recognizing it as one of the most valuable things that has come to us in late years, but explaining that it has an element of danger. We have emphasized this because the exponents of this marvelous anesthetic agent, and those who market it, make almost no mention of its toxic properties. We could wish these gentlemen who so well exploit the anesthetic, would exploit as well that there is an element of real danger, and, at the time of so doing, teach the professions how to recognize and combat that danger.

The dentist who is responsible for the narrow escape I have detailed, with fine spirit, reported all of the circumstances before the local dental society, with the result that those dentists were incredulous, and suggested that hysteria explained the symptoms. They stated how they, one and all, used novocain daily, and none of them had ever noticed the toxic effects.

The recent free discussion of a paper on local anesthesia by a country society emphasized the universal use of this method of anesthesia by these country doctors; but they had nothing to say about the possible toxic effects, either being ignorant of, or ignoring, the fact that they might occur. If we are going to be right about this question we must emphasize these important facts relative to novocain and its allied drugs for the education of the great mass of doctors and dentists. It is inconceivable that a professional man would repudiate any valuable agent merely because it has an element of danger. It is equally inconceivable that he would exhibit

that agent without knowing the possible dangers, and their symptoms and how to combat them. This is the situation as I see it.

I am asked how to combat these toxic effects, and will say that we found this patient who was suffering from them still sitting up in the dentist's chair. They had not loosened her clothing, they had not dropped her head, and, although her paralysis was obviously respiratory and not circulatory, they had done nothing, notwithstanding the fact that they had oxygen available as well as respiratory stimulants.

First of all, drop the head low, put the patient prone, give oxygen if available, loosen the clothing, especially the stays, resort to artificial respiration or, better still, use a pulmotor or lung-motor. Give respiratory stimulants by hypodermic, for, as I understand it, it is a paralysis of respiration, due to the influence of the drug on the floor of the fourth ventricle, after it has been picked up too rapidly by the blood-current, probably from an injection directly into a vein.

In making your injection, if possible avoid the veins, introduce your needle, first detached from the syringe, and notice whether any blood is coming through the needle. Move your needle back and forth slightly, after you have introduced it, prior to connecting up your syringe. These things are the simple things that any one can do.

If the gentlemen here who are to close the discussion can throw any further light upon it, I, for one, will be personally grateful.

Dr. J. Frank Corbett (Minneapolis, Minn.): I wish to make a few corroboratory statements of the testimony that Dr. Law has offered.

In the experimental laboratory, I find that when we inject novocain directly into the jugular vein, we almost invariably have some respiratory disturbance, and in a few cases cessation of breathing. Artificial respiration and nitroglycerin in one case were necessary to restore this function, although in other instances a spontaneous recovery took place. These cases of spontaneous recovery were animals under ether anesthesia.

If I may be permitted to go back to the discussion of my own paper, I wish to say that I am glad that Dr. Harris raised the point that he did. I do not wish to be understood as saying that exhaustion of the suprarenal gland is all there is to shock; but, rather, to emphasize the importance of this

factor. Shock is a very complex subject. The fact that there are no less than five or six theories of shock recognized at the present time, emphasizes this statement. We have Henderson's hyperpnea, Boise's cardiac spasm, Meltzer's inhibition, Cobbet's oligemia, and Crile's exhaustion and acidosis, to explain shock. Specific cases can be found to illustrate each of these types.

In regard to traumatic shock: I think we are not as yet in a position to make dogmatic statements. For instance, when we traumatize the peritoneal cavity of a dog, we have a great deal of trouble in producing shock. When we touch certain centers, preëminently in the hepatic region, we may get a sudden fall of blood-pressure, which is probably only equivalent to vagus stimulation, as branches of the vagus freely communicate with the hepatic plexus. Trauma of the peritoneum in other localities first raises the blood-pressure. It is only after very long-continued traumatization that we get a fall; and with this fall of blood-pressure we usually find the adrenal not exhausted; we find the splanchnic vessels full of blood, and a high specific gravity of that fluid. Therefore, one might be justified in referring to the fall of blood-pressure in these cases as a result of hemorrhage into the tissues or of concentration of the blood.

Dr. Lyon and myself are preparing an article on this subject. We have estimated the blood in both the bowel and the mesenteric vessels, and we find that in this so-called traumatic shock, the mesenteric blood is increased and the visceral blood is diminished. I think trauma of the peritoneal cavity has a somewhat doubtful place in the etiology of shock. On the other hand, when we have an individual whose vital forces become exhausted through some other agent, be that anesthesia, jaundice, or infection, and then add trauma, we get a fall of blood-pressure and exhaustion of the adrenals, which might be called shock.

Dr. Donald Macrae, Jr. (Council Bluffs, Iowa): I witnessed a number of operations, and was impressed by the discussion of Mr. Von Eisenberg relative to post-operative pneumonia in cases submitted to local anesthesia.

He made the remarkable statement to us that the post-operative pneumonias, so called, were as frequent after the local anesthesia as with the general anesthetic.

He went on to tell us that it was due to the fact that these cases would not cough up the secretions that normally are

thrown out by the ordinary individual, and also to tell us that it was their practice to instruct the nurses to insist upon these patients coughing after operation, that the post-operative pneumonias are not due to ether or chloroform, but to the fact that when the patients coughed it caused so much pain that they refused to cough enough, that is, they refused to eliminate the very thing that produced the pneumonia, and that pneumonia following the local anesthesia is just as common as with the general anesthesia.

I had the privilege and the opportunity of having to submit to an operation for hernia, performed by Dr. Harris. I got off the table two hours later, and was enthusiastic with my friends in the operating-room and in the room in which I was afterwards placed, and I said, "Why, there is nothing to it; no more general anesthesia for me." I had a great big dinner, and was happy as a lark.

An hour later I began to have pain and I suffered the most excruciating pain that any man on earth ever had. The only thing I want to say in this discussion is, that if Dr. Harris is going to do this thing, he ought to give morphine hypodermically after the operation, because Harris does not believe in it, you know; in fact, I had to call the nurse up, and tell her that if she did not call Dr. Harris up and get him to give me a hypodermic of morphine I was going to go down the fire-escape on the outside of the building, and that was all there was to it. She called up Harris, and he said: "Well, give him a hypodermic."

Dr. W. W. Grant (Denver, Colo.): No matter what the varying theories of shock are,—whether we accept Crile's vasomotor paralysis, or Henderson's deficient carbon dioxide content of the blood, or Janeway's loss of blood and peripheral irritation and exhaustion,—we know the final effect on the nerve-centers, and I do not think it is safe to refine too greatly the question as to whether the loss of blood produces "collapse" or shock. Practically, it seems to me about the same.

I admit that you may increase the degree of shock by trauma, and the theory of Dr. Crile is, that, when you cut off the brain centers by nerve-blocking, there is no cognation on the part of the brain of what you are doing or of sensation,—in other words, you relieve the patient of nerve-trauma, and therefore there will be no shock.

We have in Denver an anesthetist who has now given

he tells me, about one hundred and forty spinal anesthesias, between the third and fourth and fourth and fifth lumbar vertebræ. He gave one for me within a week in a case of senile gangrene from chronic obliterative endarteritis in a man seventy-one years of age. He used the preparation known as stovain. In the administration, the patient is kept in a position of about forty-five degrees, and in four minutes from the start I commenced the amputation of the foot. The old gentleman talked through the whole performance, and did not suffer the slightest pain nor have the slightest shock. I could not observe an hour afterwards that there was any effect or that any impression had been made on his heart. He had no pain and no stomach symptoms, or any of the ordinary after-effects.

I do not know myself personally the difference between stovain and novocain, but stovain, it is claimed, is a heavier liquid, consequently it is better to use. At any rate, these drugs should be used with every precaution. Experimentation is necessary to determine the relative value of these different drugs.

I know that cocaine is not advocated because it is considered five to seven times more dangerous than novocain. I always use a little adrenalin with the novocain in local anesthesia; and, as Dr. Harris suggests, it should never be prepared in advance because the mixture decomposes rapidly. I have never seen the slightest disadvantage in its use; but rather an advantage in the addition of the adrenalin to the novocain solution.

Dr. F. A. Dunsmoor (Minneapolis, Minn.): I am absolutely certain, convinced by experiments made on frogs, that the inhibition of the splanchnic system can be made so complete as to allow all the contents of the blood-vessels of the body to be emptied into the abdominal cavity; and, as in the case which Dr. Law reported, it would be quite possible for a person to bleed to death without having a vein opened, or any blood to escape from the body if the patient is maintained in an upright position.

I also know that this same condition may happen as the result of pain, because pain in itself is sufficient to inhibit vasomotor activity. Either pain or fear is competent to produce the same result.

We know, as the doctor has said, that the impulses received by the brain through the nervous system are made manifest

on the circulatory system always, and I think we get externally and internally, hemorrhage, equivalent to hemorrhage in shock. You can shorten the time tremendously by position. The nearer you invert the patient, the more you shorten the time.

There are plenty of people who go down through shock, of course, in varying degrees; but I know that we do them a great deal of good by substituting gravity for the loss of vasomotor power until we can get the nerve-supply back to its normal tension.

Dr. M. L. Harris (Chicago, Ill.): In regard to Dr. Macrae's question as to pneumonia: In the three hundred and fifty major operations which I have had there has been no case of pneumonia. I do not think I ever had three hundred and fifty cases of operations under ether anesthesia without several cases of pneumonia, of varying degrees.

It is a fact that the pneumonia is due to the inability to expectorate on account of pain, in other words, there is an infection by reason of inability to clear the lungs. Under ether anesthesia the amount of mucus that accumulates in the lungs is infinitely greater than under local anesthesia; and it becomes sticky and is more difficult to expectorate, therefore we have more pneumonia under ether anesthesia than we have under local anesthesia.

Dr. Macrae did have some after-pain. I think he had pain, just as he said today. But if we had put him under general anesthesia he would have had the after-pain just the same. He had no pain from the operation—had you, doctor?

Dr. Macrae: Not a bit.

Dr. Harris: After the effects of the local anesthesia worked off, then came on a burning pain. Local anesthesia has nothing to do with it. Local anesthesia does not prevent after-pain entirely, but I do not think in hernia operations 10 per cent of the patients will require anything for the pain.

I am not averse to giving morphine when indicated. I think perhaps it would be a good plan to give all patients a hypodermic after the operation, or before, to benumb the central sense-organs. I give morphine and atropin. I have stopped the morphine and scopolamin for the reason that I had so many patients sick from the scopolamin.

I have tried veronol, giving it early in the morning before the operation; but it does not benumb the sensory receptives

sufficiently. In my experience, it is not as effective as a hypodermic of morphine.

Dr. John P. Lord (Omaha, Neb.): We did not get accurately the method of use of the magnesium chloride preparations, the parts to be used in standard solutions.

Dr. Harris: Ordinarily, three parts of the novocain solution and one part calcium chloride solution. This is the best solution that I have found. It acts more quickly, and has more prolonged action, but I have faith that we will some day, and not very remotely, discover a non-toxic substance or fluid which will produce blocking, and will be better than anything we have today.

There is no question but what the novocain, in the case mentioned by Dr. Law, was injected directly into a vein. You will remember that I said the toxic effect of the novocain depends directly on the rapidity with which it is absorbed. If you inject it directly into the vein it is absorbed of course immediately; and that is why you get such toxic effects. When it goes to the nerve-center it paralyzes the nerve-center just the same as it blocks the nerve.

A very careful technic should be followed in injecting the fluid.

I never put a syringe with novocain into any tissue and inject it. I always put it in with the needle detached. I think it is very important to be exceedingly careful in the technic in all these injections, so that these accidents will not happen.

If you are injecting into the sacral canal, for instance, never put the needle on a syringe into the canal and inject it, because you might inject the fluid into some of the large veins which always surround the spinal canal; and you will immediately get the severe toxic effect of the fluid. Always put the needle in detached, and after you get it in allow it to remain there a few seconds until you are perfectly sure that you have not got it into the spinal sac, which descends as low as between the second and third sacral segments. Two or three times after putting the needle in, I have had bleeding, showing that the end of it was in a vein. We must be careful. If blood flows never inject but change the location of the needle. That is why I never inject it locally about hemorrhoids.

We have been very fortunate so far, and have never seen any bad results.

Dr. Grant: I did not know that Dr. Parsons, to whom I alluded, was here when I made the statement quoting him. He says my statement was correct, and he wants, too, the statement made that for this condition of shock from cocaine or novocain or stovain the inhalation of ether arrests it very promptly. He says this is based upon his experience, and from some observation besides concerning the use of these local anesthetics.

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St. Paul

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Ricketts, B. M.

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Yates, John L.

Oshkosh

Connell, F. Gregory

Wausau

Smith, Joseph F.

CANADA

Winnipeg

MacLean, Neil John

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